This is a digital copy of a book that was preserved for generations on library shelves before it was carefully scanned by Google as part of a project to make the world's books discoverable online.

It has survived long enough for the copyright to expire and the book to enter the public domain. A public domain book is one that was never subject to copyright or whose legal copyright term has expired. Whether a book is in the public domain may vary country to country. Public domain books are our gateways to the past, representing a wealth of history, culture and knowledge that's often difficult to discover.

Marks, notations and other marginalia present in the original volume will appear in this file - a reminder of this book's long journey from the publisher to a library and finally to you.

Usage guidelines

Google is proud to partner with libraries to digitize public domain materials and make them widely accessible. Public domain books belong to the public and we are merely their custodians. Nevertheless, this work is expensive, so in order to keep providing this resource, we have taken steps to prevent abuse by commercial parties, including placing technical restrictions on automated querying.

We also ask that you:

+ *Make non-commercial use of the files* We designed Google Book Search for use by individuals, and we request that you use these files for personal, non-commercial purposes.

+ *Refrain from automated querying* Do not send automated queries of any sort to Google's system: If you are conducting research on machine translation, optical character recognition or other areas where access to a large amount of text is helpful, please contact us. We encourage the use of public domain materials for these purposes and may be able to help.

+ *Maintain attribution* The Google "watermark" you see on each file is essential for informing people about this project and helping them find additional materials through Google Book Search. Please do not remove it.

+ *Keep it legal* Whatever your use, remember that you are responsible for ensuring that what you are doing is legal. Do not assume that just because we believe a book is in the public domain for users in the United States, that the work is also in the public domain for users in other countries. Whether a book is still in copyright varies from country to country, and we can't offer guidance on whether any specific use of any specific book is allowed. Please do not assume that a book's appearance in Google Book Search means it can be used in any manner anywhere in the world. Copyright infringement liability can be quite severe.

About Google Book Search

Google's mission is to organize the world's information and to make it universally accessible and useful. Google Book Search helps readers discover the world's books while helping authors and publishers reach new audiences. You can search through the full text of this book on the web at `http://books.google.com/`

ers' Question Compends

SSENTIALS OF
EDICAL DIAGNOSIS
COHEN & ESHNER

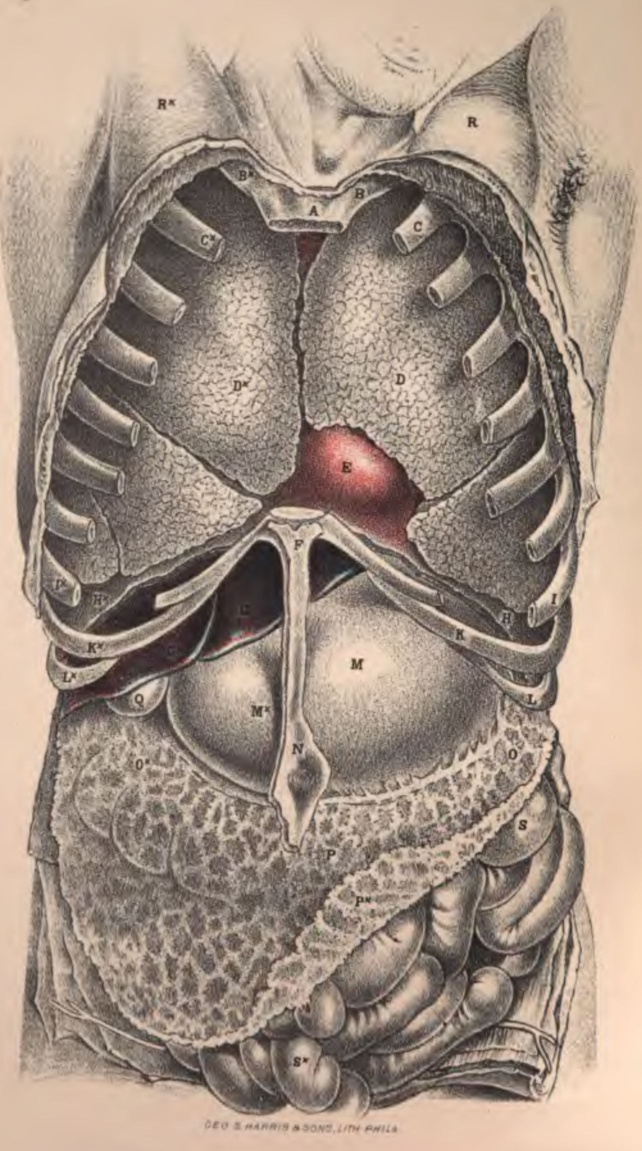
LANE
MEDICAL LIBRARY
SEAL OF THE LELAND STANFORD JUNIOR UNIVERSITY
SEMPER VIRENS
1885

Gift of

Dr. Henry L. Parish.

AMERICAN BANK NOTE CO LITHO

FRONTISPIECE.



GEO S. HARRIS & SONS, LITH PHILA.

THE RELATIONS OF THE THORACIC AND ABDOMINAL VISCERA,

THE ANTERIOR THORACIC AND ABDOMINAL WALLS REMOVED.

A.—Upper bone of the sternum.

B.B.*—Two first ribs.

C.C.*—Second pair of ribs.

D.D.*—Right and left lungs.

E.—Pericardium, enveloping the heart—the right ventricle.

F.—Lower extremity of the sternum.

G.G.*—Lobes of the liver.

H.H.*—Right and left halves of the diaphragm, in section; the right half separating the right lung from the liver, the left half separating the left lung from the cardiac extremity of the stomach.

I.I.*—Eighth pair of ribs.

K.K.*—Ninth pair of ribs.

L.L.*—Tenth pair of ribs.

M.M.*—The stomach.

N.—The umbilicus.

O.O.*—The transverse colon.

P.P.*—The omentum, covering the transverse colon and small intestines.

Q.—The gall-bladder.

R.R.*—The right and left pectoral prominences.

S.S.*—The small intestines.

SAUNDERS' QUESTION COMPENDS, NO. 17.

ESSENTIALS OF DIAGNOSIS

ARRANGED IN THE FORM OF

QUESTIONS AND ANSWERS

PREPARED ESPECIALLY FOR

STUDENTS OF MEDICINE.

BY
SOLOMON SOLIS-COHEN, M.D.,
PROFESSOR OF CLINICAL MEDICINE AND APPLIED THERAPEUTICS IN THE
PHILADELPHIA POLYCLINIC; ONE OF THE PHYSICIANS TO THE
PHILADELPHIA HOSPITAL, ETC.,
AND
AUGUSTUS A. ESHNER, M.D.,
INSTRUCTOR IN CLINICAL MEDICINE IN JEFFERSON MEDICAL COLLEGE AND IN
THE PHILADELPHIA POLYCLINIC; REGISTRAR IN THE NEUROLOGICAL
DEPARTMENT OF THE PHILADELPHIA HOSPITAL, ETC.

WITH FIFTY-FIVE ILLUSTRATIONS,
SOME OF WHICH ARE COLORED, AND A FRONTISPIECE.

PHILADELPHIA:
W. B. SAUNDERS,
913 WALNUT STREET.
1892.

Copyright, 1892.
By W. B. SAUNDERS.

TO

J. M. DA COSTA, M.D., LL.D.,

EMERITUS PROFESSOR OF THE PRACTICE OF MEDICINE AND OF CLINICAL MEDICINE
IN JEFFERSON MEDICAL COLLEGE,

THE MASTER DIAGNOSTICIAN,

THE BRILLIANT TEACHER, THE DISTINGUISHED CLINICIAN,

THIS LITTLE BOOK

IS AFFECTIONATELY INSCRIBED

BY

TWO GRATEFUL PUPILS.

88976

— 1 —
367
1892

PREFACE.

THIS book is intended to meet a popular demand. While the endeavor has been to make it reliable and helpful, the student is advised not to depend upon it to the exclusion of standard and more elaborate works. It is elementary in character, devoid of detail, and represents but an outline of the subject with which it has to deal. This outline must be filled from observation and further reading.

Being written especially for students, everything has been sacrificed to accuracy and brevity; references to authorities consulted have been omitted; and it has been deemed wisest to conform with prevailing views in matters concerning which there may be differences of professional opinion.

In the arrangement of material, systematic classification has often been departed from, to secure the benefit of association of ideas.

Diagnosis must be studied from patients, not from books; even the best of books can only direct the student what to look for at the bedside, and warn him against probable errors.

If this book facilitates the true methods of study, it will have accomplished its purpose.

CONTENTS.

PAGE
Introduction 17
Fever 27
Simple Continued Fever—Ephemeral Fever—Febricula . 31
Ardent Fever 31
Thermic Fever—Insolation—Sunstroke 32
Catarrhal Fever — Influenza — Epidemic Catarrh — La Grippe 32
Typhoid Fever—Enteric Fever 35
Typhus Fever 43
Cerebro-spinal Fever—Epidemic Cerebro-spinal Meningitis 45
Asiatic Cholera—Cholera Infectiosa 49
Relapsing Fever 51
Malarial Diseases 52
Yellow Fever 59
Weil's Disease 62
The Exanthemata 63
  Morbilli—Measles—Rubeola 63
  Scarlatina—Scarlet Fever 65
  Rötheln—Roseola—German Measles—French Measles 68
  Variola—Smallpox 69
  Varicella—Chickenpox 73
  Erysipelas 74
Dengue—Break-bone Fever 76
Diphtheria 77
Glanders—Farcy—Equinia 80
Anthrax—Wool-sorters' Disease — Charbon — Malignant Pustule—Splenic Fever 81
Actinomycosis 83

PAGE
Foot-and-Mouth Disease 84
Hydatid Disease 84
Trichiniasis 85
Acute Rheumatism—Rheumatic Fever 87
Gonorrheal Synovitis 88
Syphilitic Arthritis 89
Subacute Rheumatism 89
Myalgia 90
Chronic Rheumatism 90
Acute Gout 91
Chronic Gout 92
Lithemia 93
Rheumatoid Arthritis—Arthritis Deformans . . . 94
The Blood 95
    Anemia 97
    Chlorosis 98
    Pernicious Anemia 98
    Leukocytosis 99
    Leukemia 99
    Pseudo-leukemia — Hodgkin's Disease — Lymphatic Anemia—Malignant Lymphoma or Lymphadenoma 101
    Infantile Pseudo-leukemic Anemia 103
    Scorbutus—Scurvy 104
    Purpura 105
    Hemophilia 106
Addison's Disease 107
Rachitis 108
Mollities Ossium 108
The Heart 109
    Inspection 109
    Palpation 110
    Percussion 110
    Auscultation 110
    Malformation 115
    Dextrocardia 115
    Functional Disturbance of the Heart 116
    Tachycardia 116

PAGE
Irritable Heart 117
Angina Pectoris 118
Hypertrophy of the Heart 119
Dilatation of the Heart 120
Valvular Disease of the Heart 121
Mitral Incompetency—Mitral Regurgitation . . 122
Mitral Obstruction 123
Aortic Obstruction 123
Aortic Incompetency—Aortic Regurgitation . . 124
Tricuspid Incompetency—Tricuspid Regurgitation . 125
Tricuspid Obstruction 125
Pulmonary Obstruction 125
Pulmonary Incompetency—Pulmonary Regurgitation 125
Acute Pericarditis 127
Acute Endocarditis 128
Mycotic Endocarditis 129
Myocarditis 130
Hydropericardium 130
Heart-clot 130
Thoracic Aneurism 131
Arterio-Capillary Fibrosis 132
Symmetrical Gangrene—Local Asphyxia—Raynaud's Disease 133
The Respiratory System 134
Coryza—Acute Nasal Catarrh 136
Hay-fever — Hay-asthma — Rag-weed Fever — Autumnal Catarrh — June-cold — Rose-cold — Idiosyncratic Coryza—Periodic Vasomotor Coryza . . 137
Acute Laryngitis 138
Edema of the Larynx 139
Acute Tuberculous Laryngitis 140
Laryngismus Stridulus 141
Laryngeal Vertigo 142
Catarrhal Croup—Spasmodic Croup 142
Membranous Croup 143
Whooping-cough—Pertussis 145

PAGE
Chronic Laryngitis 146
Chronic Tuberculosis of the Larynx 147
Physical Diagnosis 147
Acute Pleurisy 153
Acute Bronchitis 157
Chronic Bronchitis 158
Plastic Bronchitis—Fibrinous Bronchitis . . . 159
Putrid Bronchitis 159
Bronchiectasis 160
Capillary Bronchitis 161
Catarrhal Pneumonia—Broncho-pneumonia—Lobular Pneumonia 162
Acute Croupous Pneumonia—Lobar Pneumonia . 163
Pulmonary Gangrene 168
Pulmonary Tuberculosis 169
Acute Miliary Tuberculosis 177
Interstitial Pneumonitis 179
Pulmonary Emphysema 179
Pneumothorax 181
Asthma 183
The Digestive System—The Mouth 184
Catarrhal Stomatitis 184
Aphthous Stomatitis 184
Thrush—Muguet—Parasitic Stomatitis . . . 185
Ulcerative Stomatitis 185
Mercurial Stomatitis 186
Gangrenous Stomatitis—Noma—Cancrum Oris . . 186
The Tongue 187
Glossitis 187
Leukoplakia Lingualis—Leukoplakia Buccalis . . 187
Glossanthrax 187
Nigritics—Black Tongue—Hairy Tongue . . . 188
Mumps—Parotiditis 188
The Pharynx 188
Pharyngitis 188
Tonsillitis 191

PAGE
Herpetic Sore-throat—Herpetic Tonsillitis—Herpes of the Pharynx—Common Membranous Sore-throat—Ulcero-membranous Angina—Diphtheroid Throat . 192
Gangrenous Pharyngitis—Putrid Sore-throat . . 193
Retro-pharyngeal Abscess 194
The Esophagus 194
Stricture of the Esophagus 194
The Stomach 195
Gastralgia 196
Acute Gastritis 197
Chronic Gastritis—Chronic Gastric Catarrh . . 198
Gastric Ulcer 199
Carcinoma of the Stomach 200
The Intestines 203
Acute Enteritis 203
Croupous Enteritis—Membranous Enteritis . . 205
Cholera Morbus—Cholera Nostras 205
Cholera Infantum 206
Chronic Enteritis—Chronic Intestinal Catarrh . . 206
Acute Dysentery 206
Chronic Dysentery 208
Typhlitis 208
Perityphlitis 209
Intestinal Obstruction 211
Intussusception—Invagination 212
Carcinoma of the Intestine 213
Intestinal Parasites 214
Tenia Solium 214
Tenia Mediocanellata 215
Bothriocephalus Latus 216
Ascaris Lumbricoides—Round-worms . . . 217
Oxyuris Vermicularis—Seat-worms—Thread-worms—Spool-worms 218
Acute Peritonitis 219
Chronic Peritonitis 221
Tabes Mesenterica 222

PAGE
The Liver 222
Floating Liver 223
Congestion of the Liver 224
Acute Hepatitis 225
Acute Yellow Atrophy of the Liver 226
Abscess of the Liver 228
Interstitial Hepatitis—Cirrhosis of the Liver . . 229
Fatty Liver 230
Amyloid Disease of the Liver 231
Carcinoma of the Liver 232
Hydatid Cyst of the Liver 234
Perihepatitis 235
Cholangitis—Cholecystitis 236
Biliary Calculi 238
The Spleen 240
Floating Spleen 241
Splenitis 241
The Pancreas 241
Acute Pancreatitis 241
The Genito-Urinary Apparatus 242
Oxaluria 248
Pyuria 249
Albuminuria 250
Chyluria 253
Lipuria 253
Hematuria 254
Hemoglobinuria 254
Paroxysmal Hemoglobinuria 255
Endemic Hematuria 256
Diabetes Insipidus 256
Diabetes Mellitus 257
Cystitis 261
Vesical Calculus 262
Neoplasms of the Bladder 262
Pyelitis 263
Renal Inadequacy 263
Floating Kidney 264

PAGE
Renal Calculus 264
Acute Nephritis 265
Chronic Parenchymatous Nephritis 265
Amyloid Kidney 266
Chronic Interstitial Nephritis 266
Abscess of the Kidney 268
Perirenal Abscess 269
Tuberculosis of the Kidney 269
Malignant Disease of the Kidney 270
Hydronephrosis 270
Hydatid Cyst of the Kidney 271
The Nervous System 272
Neuritis 275
Multiple Neuritis 276
Sciatica 278
Paralysis of the Facial Nerve 279
Paralysis of the Phrenic Nerve 281
Paralysis of the Musculo-Spiral Nerve . . . 281
Neuromata 282
Neuralgia 283
Migraine 284
Spinal Meningitis 285
Cervical Pachymeningitis 286
Hemorrhage into the Spinal Membranes . . . 286
Anemia of the Spinal Cord 287
Hyperemia of the Spinal Cord 287
Myelitis 288
Chronic Myelitis 289
Acute Anterior Poliomyelitis 290
Acute Ascending Paralysis 291
Amyotrophic Lateral Sclerosis—Progressive Muscular Atrophy—Glosso-labio-laryngeal Palsy . . . 292
Acute Bulbar Palsy 296
Pseudo-bulbar Palsy 296
Pseudo-hypertrophic Paralysis 296
Simple Idiopathic Muscular Atrophy 298
Arthritic Muscular Atrophy 299

PAGE
Thomsen's Disease—Myotonia Congenita . . . 300
Posterior Spinal Sclerosis—Locomotor Ataxia—Tabes Dorsalis 300
Primary Lateral Sclerosis—Spastic Paraplegia . . 302
Postero-lateral Sclerosis—Ataxic Paraplegia . . 303
Friedreich's Ataxia—Hereditary Ataxic Paraplegia . 304
Cerebro-spinal Sclerosis—Insular Sclerosis—Multiple Sclerosis—Disseminated Sclerosis 305
Paralysis Agitans—Shaking Palsy 306
Spinal Hemorrhage 308
Spinal Compression 309
Tumor of the Spinal Cord 310
Syringomyelia 312
Morvan's Disease—Analgesic Panaris . . . 312
Cerebral Meningitis 315
Hydrocephalus 318
Hemorrhage into the Cerebral Membranes . . . 318
Congenital Spastic Paraplegia 319
Cerebral Anemia 320
Cerebral Hyperemia 321
Cerebritis 321
Cerebral Abscess 322
Cerebral Hemorrhage 323
Cerebral Softening 327
Cerebral Embolism 328
Cerebral Thrombosis 329
Cerebral Tumor 330
Intracranial Aneurism 331
General Paralysis of the Insane—Paretic Dementia . 332
Sunstroke — Heat-stroke — Insolation — Heat-fever — Thermic-fever—Heat-exhaustion 334
Delirium Tremens 335
Plumbism 337
Torticollis 338
Occupation-Neuroses 339
Writer's Cramp 339
Chorea 340

PAGE
Epilepsy 341
Hysteria 344
Tetanus 347
Tetany 349
Hydrophobia 350
Aural Vertigo—Labyrinthine Vertigo—Menière's Disease 351
Exophthalmic Goiter—Graves's Disease—Basedow's Disease 351
Cretinism 354
Myxedema 354
Akromegalia 355

LIST OF ILLUSTRATIONS.

FRONTISPIECE.

FIG. PAGE

1. Bacilli of Typhoid Fever (von Jaksch) . . . 36
2. Temperature-chart of a case of Typhoid Fever (Wunderlich.) 37
3. Temperature-chart of a relapse in Typhoid Fever. Convalescence from the relapse interrupted by recrudescence 39
4. Temperature-chart of a case of Pyemia (Wunderlich) 41
5. Comma-bacillus of Cholera (Vierordt) . . . 49
6. Spirochetæ of Relapsing Fever (v. Jaksch) . . 51
7. Organisms of Malaria—Intracorpuscular (Macé) . 53
8. Organisms of Malaria—Extracorpuscular (Macé) . 53
9. Temperature-chart of Quotidian Intermittent Fever (Wunderlich) 55
10. Temperature-chart of Tertian Intermittent Fever (Wunderlich) 55
11. Temperature-chart of Measles (Strümpell) . . . 64
12. Temperature-chart of Scarlatina (Strümpell) . . 66
13. Temperature-chart of Smallpox (Strümpell) . . 69
14. Anthrax-bacilli in Blood (Vierordt) 82
15. Actinomyces (Ziegler) 83
16. Tenia Echinococcus—Vesicle, Scolex and Hooks (after Heller) 85
17. Trichina (v. Jaksch) 86
18. Appearance of the Blood in Leukemia (Funke) . . 100
19. Auscultation of the Heart-sounds (Vierordt) . . 113
20. Normal Pulse-tracing (after Eichhorst) . . . 115

FIG. PAGE
21. Pulse-tracing of Aortic Insufficiency (after Strümpell) 124
22. Micrococci of Croupous Pneumonia (Vierordt) . . 164
23. Tubercle-bacilli in Sputum (Ziegler) 173
24. Tenia Solium, magnified (Heller) 215
25. Tenia mediocanellata, magnified (Heller) . . . 216
26. Bothriocephalus Latus, magnified (Heller) . . . 216
27. Ascaris Lumbricoides (v. Jaksch) 217
28. Oxyuris Vermicularis, natural size (Vierordt) . . 218
29. The Relations of the Heart, Lungs, Liver, Stomach and Spleen, as seen from the front (Weil) . . 223
30. Showing the Relations of the Lungs, Liver, Spleen and Kidneys, as seen from behind (Weil-Luschka) . 224
31. Relations of the Spleen (Weil) 240
32. Uric Acid and Urates (Funke) 244
33. Calcium Phosphate (Laache) 245
34. Triple Phosphates and Ammonium Urate (Laache) . 246
35. Leucin and Tyrosin (Laache) 247
36. Calcium Oxalate (Laache) 248
37. Red Blood-corpuscles and a Blood-cast of a Uriniferous Tubule (Eichhorst) 251
38. Epithelial Cast of a Uriniferous Tubule (v. Jaksch) . 251
39. Granular Casts of the Uriniferous Tubules (v. Jaksch) 251
40. Hyaline Casts of the Uriniferous Tubules (Vierordt) . 251
41. Waxy Casts of the Uriniferous Tubules (v. Jaksch) . 252
42. Filaria Sanguinis Hominis (v. Jaksch) . . . 253
43. Multiple Neuritis—Wrist-drop and Foot-drop (Gowers) 277
44. Facial Paralysis (Gowers) 280
45. Progressive Muscular Atrophy (Gowers) . . . 294
46. Pseudo-hypertrophic Paralysis in two brothers (Gowers) 297
47. Pseudo-hypertrophic Paralysis. Mode of rising from the ground (Gowers) 298
48. Paralysis Agitans (after St. Leger) 307
49. Appearance of the Hand in Morvan's Disease (after Charcot) 313
50. Cerebral Miliary Aneurisms (Eichhorst) . . . 324
51. Wrist-drop from Lead-poisoning (Gowers) . . . 337

FIG. PAGE
52. Mode of holding the pen favorable to the development of Writers' Cramp (Gowers) 340
Method of holding the pen when writing becomes difficult (Gowers) 340
53. Exophthalmic Goiter. Defective descent of the upper lids on looking down (after Wilks) 352
54. Akromegalia (after Marie) 356
55. Outline of the Face: 1, In Myxedema; 2, In Akromegalia; 3, In Osteitis Deformans (Marie) . . 357

ESSENTIALS OF DIAGNOSIS.

INTRODUCTION.

DIAGNOSIS, in medicine, is the art and science of observing and of discriminatingly interpreting the phenomena of disease. In its study, a knowledge of the phenomena of health is an essential prerequisite. Unless familiar with the sounds elicited upon percussion of the normal chest, one cannot decide whether or not the sound heard in a given case is indicative of abnormal conditions. Unless one knows the characteristics of normal urine, he cannot hope to gain from urinalysis a clue as to the nature of a case of disease. Unless one knows the appearance of a healthy brain, he cannot determine whether the brain seen at a necropsy is or is not the seat of morbid change. Unless one knows the function of a normal joint, he cannot affirm that a joint under investigation has had its function impaired.

Having learned, by observations upon the healthy, to recognize when departures from health have taken place, one must further learn by observation of the sick to appreciate the significance of such departures. No opportunity should be lost to examine *post mortem* the organs and tissues that have been altered by disease.

A knowledge of the effects of drugs upon healthy and unhealthy persons and organs may likewise render easy a diagnosis that might otherwise be difficult.

The phenomena indicative of the existence of disease are in a general way termed *symptoms*. These may be either *subjective*—known only to the patient by his sensations; or they may be *objective*—capable of investigation by the senses of the observer, aided, it may be, by instruments of precision. Thus pain, vertigo, nausea, ringing in the ears, are subjective symptoms, while high temperature, vomiting, unsteadiness of motion, loss of voice, are objective symptoms.

The objective manifestations of disease may be further divided into *symptoms* (in a restricted sense) and *signs*. While the two are not rigidly separable, *symptoms* may be defined as manifestations of disordered function, *signs* as manifestations of altered structure. Thus, cough is a symptom; the laryngeal congestion discovered by inspection with the mirror, the bronchial râles heard upon auscultation, are signs. Difficulty in deglutition is a symptom; a pulsating tumor in the chest, indicative of aneurism pressing upon the esophagus, is a sign. Dyspnea is a symptom; distension of the abdomen with gas (tympanites) or with fluid (ascites), causing pressure upon the diaphragm and restricting its movements, is a sign.

Thus, signs are in many cases directly explanatory of symptoms, and their discovery is one step further in the diagnosis. It must not be forgotten, however, that signs are not always to be found; that, when found, a sign may not account *in toto* for the symptom with which it is associated; and that even when sufficient to explain the symptom, the sign itself remains to be explained before the diagnosis is complete. Like all other morbid phenomena, signs must, therefore, be considered in relation with all the evidence presented in a given case.

Certain signs that are not at once manifest, but that require for their study special means of exploration, are termed *physical signs*. In its restricted sense, the term "physical signs" is applied to the phenomena elicited by special methods (*inspection*, *mensuration*, *palpation*, *percussion*, and *auscultation*), used chiefly in examination of the chest and abdomen, though often applied elsewhere; while the signs elicited by examination of the blood, the urine, the sputum, the feces, or by laryngoscopy, ophthalmoscopy, cystoscopy and the like, are not given other qualifying designation than the special names describing the respective processes.

Phenomena corresponding with logical deductions as to the direct results of deranged function, including subjective and objective symptoms and signs, are called *rational signs*, in contradistinction from physical signs, which denote the mechanical condition of the structures examined. Thus, in a case of valvular disease of the heart, weakness, vertigo, shortness of breath, pallor and dropsy would be termed rational signs, while the area of cardiac percussion-dulness and the character of the sounds heard upon auscultation would be termed physical signs.

Symptoms may also be divided into *general* or *constitutional* symptoms and *local* symptoms. General symptoms are those, like fever, depression, delirium, that may result from unbalancing of the organism as a whole, and are common to affections of many kinds; while local symptoms are those, like swelling or discoloration of a part and circumscribed pain or tenderness, that result from localized morbid conditions and are confined to a certain locality, usually that of the organ or tissue diseased.

Symptoms caused by local disease not at the seat of manifestation and that are not dependent upon mere mechanical influences or upon interference with related function, but that arise indirectly, as a result of nervous irritation, are termed *reflex symptoms*. Thus, the difficulty of breathing occasioned by the pressure of a mediastinal tumor upon the trachea is a local symptom, due to a mere mechanical influence; dyspnea from deranged action of the heart is a symptom dependent upon interference with related function; while an asthmatoid condition dependent upon disease of the nose is a reflex symptom. It must not be forgotten, however, that the same symptom may at one time be local; at another time, part of a general process; at another time, reflex. Thus, vomiting may indicate local disease of the stomach; or it may be part of the general disturbance caused by certain febrile diseases; or it may reflexly indicate disease in the abdomen or in the brain.

Having, by careful observation and interrogation, ascertained the *present condition* of the patient and having, by inquiry, learned his *family history* (in order to judge of the probable influence of hereditary disease, diathesis or liability) and his *previous history* (anamnesis) of health or disease (including a knowledge of his mode of life and of his surroundings, as well as the mode of invasion of the disease under investigation and its course up to the moment of examination), it becomes necessary to interpret the information thus gained—in other words, to *make a diagnosis.*

In making a diagnosis one has to consider not only the bearing of the signs and symptoms individually and collectively, but also their relations with the phenomena of health and with each other. Further, one has to consider (and this is what may, to some extent, be learned from reading) the historical experience of the medical profession as to the significance of certain symptoms and groups of symptoms, and as to the relation of certain symptoms and groups of symptoms with lesions observed *post mortem.*

A diagnosis may thus be made (1) by the *inductive method,* reasoning, upon anatomical (structural) and physiological (functional) data, from the character of the disturbance to the organ affected and the nature of the affection ; (2) by the *historical* or *empirical* method, relying upon the recorded experience of other observers and upon one's own experience that certain symptoms manifested under certain circumstances indicate the existence of a definite malady ; or (3) by the method of *pathological association*, which is based upon the fact that when certain symptoms have been observed during life, definite lesions have been discovered after death. By analogy, the lessons of pathological association may be applied in affections not necessarily of a fatal character. It is obvious, therefore, that a knowledge of the various gross and minute morbid changes occurring in the body generally or in special organs, tissues or cells and of the circumstances under which special changes are likely to occur is essential for precision in diagnosis. The most satisfactory results are to be obtained when all of the methods indicated can be concurrently availed of.

Diagnosis may further be *direct*, *differential* or by *exclusion.* Diagnosis is said to be *direct* when one or more of the signs or symptoms, independently of or in relation with other symptoms or with the age, sex, physical and mental characteristics, residence or occupation of the patient, or with his family history, enable direct affirmation to be made of the nature of the malady. Thus, a paroxysm of chill, fever and sweating, in association with the presence in the blood of characteristic parasites, permits a direct diagnosis of malarial fever to be made.

Diagnosis is said to be *discriminative* or *differential* when the signs or symptoms are suggestive of more than one disease and a decision is reached by comparison and contrast. It is necessary to compare the ideal pictures of various diseases in turn with the actual picture presented, in order to establish the resemblance or unlikeness; finally affirming the nature of the case with more or less certainty, according to the completeness and definiteness of the observations made and the degree of knowledge on the part of the clinician as to the conditions present in the respective affections under review. Diagnosis is usually differential, and as one is often compelled to balance probabilities, with incomplete evidence before him, differential diagnosis calls for the greatest knowledge and skill. Numerous examples of differential diagnosis will be found throughout this book. Reference may here be made to malarial fever, in case hematozoa are not demonstrable, and the fever of hepatic suppuration or of pulmonary tuberculosis. Careful observation of the temperature-course and painstaking physical examination may be required to establish the points of difference.

In diagnosis by *exclusion* one is unable to affirm the nature of the affection directly; and even after comparison of the evident phenomena with the phenomena of the respective diseases suggested, the points of resemblance are not sufficiently great in number or in character to warrant an affirmative conclusion in any one instance. It then becomes necessary to prove a negative; to do which, reliance must be placed entirely upon points of unlikeness. One endeavors to recall in the ideal picture of a certain malady some symptom or association of symptoms so necessary that its absence from the actual case may warrant a decided negative; or, on the other hand, to discover in the case before him some symptom or association of symptoms so incompatible with the ideal picture of the malady under consideration as likewise to warrant a negative conclusion. Thus, from the absence of what ought to be present and from the presence of what ought to be absent, one after another of the conditions discussed is set aside, until finally one remains that cannot be so excluded; and there is reached a more or less probable diagnosis.

Diagnosis by exclusion is the least satisfactory, as one can never be sure that he has passed in review and has excluded all of the conditions that ought to be excluded. Nevertheless, it sometimes affords most brilliant results. Thus, paralysis of the left vocal band, associated with recurring cough and occasional dyspnea and dysphagia, every other suggested cause for which has been excluded, has led to a correct diagnosis of aneurism of the arch of the aorta, not discoverable by the most careful physical exploration.

As a rule, that diagnosis that best and most readily accounts for all of the symptoms is the most likely to be correct. When any symptom is unaccounted for, the diagnosis is at least incomplete, if not doubtful. Diagnosis of a common disease, other things being equal, is more likely to be correct than diagnosis of a rare disease. On the other hand, statistics have no bearing upon the individual case; the rare disease may be present and be overlooked, from want of knowledge or in consequence of superficial examination.

More than one disease may be present in the same patient at the same time; but certain diseases are held to be antagonistic and thus not likely to coëxist. Data bearing on this point are defective and are undergoing revision and correction. Not a few cases have been recorded contradictory of some of the teachings of the past. Hence, in this work, little stress has been laid upon these antagonisms. On the other hand, there are many lesions and morbid processes that are frequently found associated. Such associations are specifically mentioned whereever it seemed likely that a knowledge of their existence would be of service.

One disease sometimes strongly predisposes to another. A knowledge of facts of this nature is often of assistance in diagnosis, as well as in preventive treatment.

A knowledge of the *complications* and *sequelæ* that ordinarily or exceptionally attend or follow certain diseases, independently of its bearing upon therapeutics, is an important equipment for the diagnostician. This can be acquired only by extensive reading or by prolonged experience. In anticipation of the latter source of information, the former must be sedulously cultivated. In default of such knowledge, the mistake may be made of diagnosticating a single well-marked condition, as, for instance, pleuritis, as the whole of the disease; when, in reality, the condition may be but a comparatively unimportant complication in the course of typhoid fever or of an hepatic abscess. Similarly, a sequela to some acute disease, remote in time or obscure in symptoms, may be diagnosticated, prognosticated and treated as an independent affection, often to the grave detriment of the patient. Thus, a temporary loss of knee-jerk following unrecognized diphtheria has led to an incorrect diagnosis of locomotor ataxia. Sometimes the initial phenomena of disease escape observation (a chancre may be concealed, especially in the female, or scarlatina may occur without eruption or appreciable fever, thermometry not being resorted to), and when the later phenomena (such as syphilitic fever or scarlatinal dropsy) develop, the case may be misinterpreted, unless this possibility of masked beginnings be borne in mind.

The so-called *negative signs* of disease should not be underrated. The absence of headache may assist in the exclusion of brain-tumor; the absence of albumin and casts from the urine, after sufficiently careful and extended observation, may be of great importance in discriminating among the causes of a train of symptoms including vertigo, optic neuritis, vomiting and paroxysmal dyspnea; the absence of rose-rash and of splenic enlargement may determine the diagnosis between enteric influenza and typhoid fever. In recording cases it is necessary to note negative points in order to show that the inquiry or search has been made.

No less important is a knowledge of the morbid phenomena that may be caused by certain drugs—not merely by the narcotic agents, opium, belladonna, and the like, but by such poisons as mercury, arsenic and lead, which may produce symptoms closely resembling acute and chronic diseases of common occurrence or may give rise to nervous and other phenomena puzzling in the extreme. Similarly, common or unusual drug-effects, perhaps due to idiosyncrasy, occurring in the course of treatment, may mask the symptoms of disease, or give rise to additional phenomena that, unless caution be exercised, may lead to error in diagnosis.

The *data* for diagnosis are obtained by *observation* and *inquiry*.

Inquiry may often have to be made of those about the patient, the latter being unable or incompetent to answer; and not rarely the same questions will have to be repeated in various ways and be controlled by questions requiring opposite answers, in order that the clinician be not deceived, intentionally or otherwise.

Observation can be made while the questioning is proceeding; sometimes the manner, method and form of a patient's answers to questions are in themselves part of the data acquired by observation. For example, in cases of aphasia, the patient is unable to find words in which to express himself, though he may understand the questions put and may even believe that in his monotonous repetition of certain words and phrases he has given an intelligent answer.

Observation includes (1) a more or less rapid survey of the *general condition* of the patient, and (2) a careful examination into the condition of *special structures*, the performance of *special functions* and the constitution, chemic and microscopic, of the *blood*, the *secretions* and the *excretions*, both as to normal and abnormal constituents.

The *whereabouts* of the patient may afford information as to the acuteness or severity of the attack. He may be attending to his work as usual, or he may be confined to his house, his room, his bed.

Position and movement are next noted. Thus, if the patient paces restlessly about, a condition of excitement, of physical or mental causation, is indicated. If, whether seated or recumbent, his position is easy and unconstrained, it indicates that, whatever the disease may be, the attack is, for the time being, mild and that there is no serious respiratory or circulatory trouble, or inflammation of an important part. If the patient lie passively upon his back, or huddled up in a heap, a prostrating sickness, usually an acute infectious disease, is indicated. If he lie persistently on one side, affection of the lung or pleura of that side is likely to exist; because this position restrains the movement of that side of the thorax, lessens the pain of acute inflammation and, in addition, permits the healthy lung to better perform its work. Sometimes, however, patients with pneumonia lie on the sound side, having less pain in that posture.

If, whether the patient be in an easy-chair, or in bed, the thorax is propped into an upright or semi-upright posture, there is great difficulty in respiration (*orthopnea*), which may be be due to cardiac or pulmonary disease, to obstruction in the upper air-passages, or to abdominal or general effusions. If the patient lie upon his back with the legs drawn up, there is likely to be peritonitis, and if on the right side with the right leg drawn up, appendicitis should be suspected.

If the head is retracted, or fixed in opisthotonos, disease of the cerebrum or of the meninges is to be suspected. So, too, deviation of the head to one or the other side, picking at the bedclothes, helplessness of one or more extremities, general restlessness. jactitation, local spasms or general convulsions, incoördination, tremor, temporary or persistent, sometimes throw considerable light on the condition of the nervous system.

The *expression of the countenance* should be observed. It may be indicative of comparative comfort or of intense suffering; it may be dull and apathetic, as in typhus fever; sunken and anxious, as in cholera; eager and brilliant, as in some cases of pulmonary tuberculosis; indeed, a careful study of the physiognomy of patients may often directly reveal much, or indirectly serve to lead the attention of the examiner to matters that might otherwise be overlooked. Thus, there is in many cases of chronic nephritis a peculiar pallor that can hardly be mistaken by the experienced observer. Connected with the expression of the face are the *general nutrition*, the condition of the *skin*, and the condition of the *mind*, all of which must be included in the general examination.

Concerning the *mental condition*, without special effort it can be learned whether the patient is interested in his surroundings or is oblivious to them, whether he is conscious or unconscious, whether he is delirious or is aware of his sayings and doings, whether he is able to fix his attention or is continually wandering. To the facts developed on special inquiry no attention is now being paid.

The *skin* may be of normal hue, pallid from impoverishment of blood, flushed with fever, discolored by inflammations, bruises, extravasations, eruptions, jaundice, the various cachexias. It may be swollen, or give evidence of swelling or effusion beneath. It may be abnormally smooth or rough, dry or moist, hot or cold.

The *state of the nutrition* is usually evident in the preservation of the full, rounded outlines, or in the sharpness and hollows of emaciation. For accuracy and completeness of knowledge the weight should be determined by the scales.

Abnormal prominences of various kinds and situation may indicate the existence of *tumors*, *effusions*, *articular*, *osseous*, *glandular* and *visceral enlargements* of inflammatory or other origin, to be determined by further examination.

Pulsations may be visible that, by their abnormal situation or abnormal character, are indicative of abnormal circulatory conditions, or of tumors or collections of fluid external to the heart or vessels. The tortuosity of visible arteries, or the localized or general turgescence of superficial veins, may indicate disease or obstruction in or affecting the circulatory apparatus.

The manner in which *breathing* is performed, the character of the *voice*, the nature of a *cough*, afford information as to the condition of the respiratory apparatus.

Examination of the *pulse*, the *tongue*, and the *temperature*, while affording general indications, yet mark the transition from general to special examinations.

Except in a few instances, the plan of this book does not contemplate descriptions of methods of special examination, or enumeration of the conditions indicated by special symptoms and signs (*semeiology*, *inductive diagnosis*), but rather an account of the observed clinical groupings of signs and symptoms in the recognized and named affections of nosology (*historical diagnosis*, *pathological association diagnosis*), and an elucidation of the points of contrast between diseases most likely to be confounded (*differential diagnosis*).

FEVER.

What is fever?

Fever is a complex morbid process, of which the most striking feature is elevation of temperature. Sometimes the term fever is restricted to the abnormal heat, but this is more correctly termed *pyrexia.* In addition to pyrexia, fever is attended with acceleration of the pulse and of the respiration, with thirst and with disturbance of digestion. There are likewise increased and perverted tissue-changes, as manifested by alterations of secretions and excretions, which may be completely arrested or diminished in quantity and changed in quality. As a result of the deprivation of the normal products of metabolism and of the retention in the blood of abnormal products of metabolism, as well as of the waste-products of normal metabolism, other symptoms are often caused, such as malaise, depression, headache, insomnia, delirium, etc. When fever is long-continued, wasting takes place; partly as a result of the abnormal heat, both directly and indirectly; partly from other causes. Many febrile processes are at some period in their course attended with subnormal temperature.

What is the average normal temperature of man?

The *average normal temperature* of the adult is 98.4° or 98.6° F. (37° C.); in health it may vary from this a little more than a degree; from 97.3° F. (36.25° C.) to 99.5° F. (37.5° C.). It is higher in children than in adults, and in the aged it is sometimes a few tenths higher or lower.

The temperature is increased after meals by the activity of digestion; it is increased by exertion, and in children and hysterical persons by emotion. Weather has but slight influence upon the temperature of the healthy; it often has a marked influence upon that of the sick. There are daily periodic variations. The *daily minimum* occurs in the early morning between two and five o'clock; the *daily maximum* is reached between five and eight o'clock in the evening. The *average difference* between maximum and minimum is about 1.5° F.; it may be much more. In the tropics the average body-heat is said to be slightly higher, and the daily range greater than in temperate climates.

In sickness the diurnal variations (morning remission, evening exacerbation) become quite marked.

What is subfebrile temperature?

The term *subfebrile temperature* is not to be confounded with *subnormal* temperature. It is applied to a moderate morbid elevation of temperature not exceeding 100.4° F. (38° C.).

What is the ordinary range of febrile temperature?

A temperature of from 100.4° F. (38° C.) to 101.2° F. (38.4° C.) is considered an indication of *slight fever*.

A temperature of from 101.3° F. (38.5° C.) to 102.2° F. (39° C.) in the morning, and of 103° F. (39.5° C.) in the evening, is indicative of *moderate fever*.

A temperature of from 103° F. (39.5° C.) to 104° F. (40° C.) in the morning, and of 105° F. (40.5° C.) in the evening, indicates *high fever*.

What is hyperpyrexia?

Febrile temperature exhibiting a tendency to remain above 107° F. (41.7° C.) is called *hyperpyrexia*. This is ordinarily directly dangerous to life.

Very high temperatures have been observed in hysterical cases in which, apparently, deception has been guarded against; a case of injury to the spine, in which recovery took place, is reported to have exhibited on many occasions a temperature of 122° F. (50° C.).

What is meant by inverse temperature?

Ordinarily the course pursued by morbid temperature is like that of normal temperature in having its maximum towards evening, and its minimum in the early morning. Sometimes, however, the maximum may be much earlier or much later than usual, even at noon or at midnight. Sometimes there are great fluctuations during day and night. When a complete reversal takes place, so that the *morning* temperature exhibits the *maxi-*

mum, and the *evening* temperature the *minimum*, the condition is said to be one of "inverse temperature."

Inverse temperature not infrequently occurs in acute tuberculous affections. It is of rather rare occurrence in typhoid fever.

What is the significance of a sudden fall of temperature?

A sudden fall of temperature may be part of the usual phenomena of a disease, as in intermittent fever; or it may indicate termination by crisis, as in pneumonia; or when it is not part of the usual course, and is not brought about by obvious loss of blood (as by venesection or menstruation), by drugs or cold applications, or other extraneous influence, it may indicate the development of albuminuria, or the occurrence of internal hemorrhage.

What is subnormal temperature?

A temperature lower than 97.3° F. (36.25° C.) is considered *subnormal*.

The temperature may be subnormal in influenza and in acute yellow atrophy of the liver as part of the essential course of the disease.

Subnormal temperature occurs, usually associated with profuse perspiration, at periods of critical recovery, as in pneumonia. There is simultaneous decrease in the frequency of pulse and of respiration and improvement in the general condition and sensations of the patient. In many acute diseases that terminate by lysis, such as typhoid fever, the temperature falls below the normal with the setting in of convalescence.

A subnormal temperature develops abruptly in conditions of collapse. It is then associated with sudden failure of the heart, as indicated by a feeble, rapid and irregular pulse, a fluttering and feeble cardiac impulse, pallor, coldness of the skin, with or without sweating, faintness or syncope, and profound prostration and weakness. The pulse becomes feebler, less rapid, and may finally cease. If recovery take place, the temperature again rises; or, if the termination be fatal, the temperature may rise *post mortem*.

The temperature becomes subnormal in some cases of severe hemorrhage, and at times, temporarily or persistently, in chronic wasting diseases, such as pulmonary tuberculosis and carcinoma, in chronic Bright's disease and in some diseases of the brain.

What is an essential fever?

An *idiopathic* or *essential fever* is one in which the pyrexia and its concomitants do not result secondarily from an anatomical lesion, but arise primarily from the action of a specific poison or a similar cause. Essential fevers may or may not present invariable lesions as a part of their phenomena.

What is a symptomatic fever?

A *symptomatic* or *deuteropathic fever* is one that arises secondarily as the result of irritation, intoxication or perverted function, caused by disease or injury of a special organ or tissue. The causative lesion is the essential element; the fever is but one of the symptoms.

What is a specific fever?

A *specific fever* is an essential fever due to a specific pathogenetic agent. Typhoid fever and yellow fever are examples of specific fevers.

What is a continued fever?

A *continued fever* is one in which the temperature pursues an uninterrupted course, without sudden variation from beginning to end. The diurnal range will not exceed 1.8° F. (1° C.). The first rise may be sudden or gradual; there may be steady maintenance of a maximum, or continuous increase; there may be gradual or sudden final defervescence; but there is not a decided fall and a renewed rise during the progress of the case.

What is a remittent fever?

A *remittent fever* is one the course of which is interrupted once or repeatedly by a marked decline in temperature, not reaching the normal, and followed by renewed exacerbation. The daily difference wil exceed 1° C. (1.8° F.).

What is an intermittent fever?

An *intermittent fever* is one in the course of which a complete intermission takes place once or repeatedly; that is to say, in the progress of the case the temperature becomes normal (or even subnormal), remains normal for an appreciable time, of greater or less duration, and subsequently rises to about its previous level. The maximum may be high.

Simple Continued Fever.

What is the clinical course of simple continued fever?

Simple continued fever, *ephemeral fever* or *febricula* is a non-specific fever, sometimes apparently idiopathic, but often symptomatic of digestive aberration or of some local irritation. It may be *caused* by error in diet, by fatigue, anxiety, shock, unusual mental or physical exertion, exposure to cold and damp to a foul atmosphere, or to the sun.

The disease *sets in* with a feeling of indisposition, which is followed by chilliness, fever, with heated skin, headache, thirst, anorexia and pain in the back and limbs. The *pulse* is accelerated. The *tongue* is coated. There is usually *constipation*, less frequently *diarrhea*. The *urine* is scanty and high-colored. The *pyrexia* is slight or moderate; the diurnal variations are small, the rise rapid, the fall gradual or by critical defervescence, with profuse sweating or watery diarrhea. The *duration* is commonly less than a week. It may be less than twenty-four hours. Recovery is invariable and convalescence is rapid.

Graver forms of the affection, induced by the graver causes, may be attended with considerable depression, and running a more protracted course, in duration from ten to fourteen days, may simulate typhoid fever. Especially is this resemblance marked in cases of septic intoxication.

Ardent Fever.

What is ardent fever?

Ardent fever is a non-specific, continued fever, occurring in hot countries, in which the ordinary symptoms of febricula are exaggerated. The *temperature* reaches or exceeds 103° F.; there is throbbing of the temporal arteries, with severe headache and even delirium. The symptoms resemble those of an inflammatory fever, so that the utmost care must be observed to avoid error in diagnosis.

Thermic Fever—Insolation—Sunstroke.

What is thermic fever?

Thermic fever, or *sunstroke*, results from exposure to extreme heat. The heat may be excessive and the exposure brief, or the heat may be less intense and the exposure prolonged. The condition is described with affections of the nervous system.

Catarrhal Fever—Influenza.

What is catarrhal fever?

Catarrhal fever, *epidemic catarrh*, *influenza* or *la grippe*, now commonly called "grip" in the United States, is an infectious, possibly contagious disease, belonging to the group of specific, essential, continued fevers, that occurs epidemically, endemically or pandemically, more rarely sporadically, and exhibits a protean capability of variation in its symptoms.

There are four prominent *varieties:* (1) the *catarrhal*, in which the predominant features are those of catarrhal inflammation of the respiratory tract; (2) the *thoracic*, in which, with or without the ordinary catarrhal manifestations, there are symptoms of profound involvement of the thoracic viscera; (3) the *abdominal* or *gastro-intestinal*, in which disturbances of the digestive tract are most marked; (4) the *nervous* or *cerebral*, in which neural phenomena attract attention, and in which, as in the abdominal variety, the clinical course of typhoid fever is often closely simulated. Much greater refinement in subdivision might be made, as the symptoms of two or more types are frequently associated in one case. Some epidemics are characterized by greater or even exclusive prevalence of one or two types; but in other epidemics, half a dozen patients in one household may exhibit six varieties of the disease.

What is the clinical course of influenza?

The *invasion* of influenza is commonly sudden, sometimes with a chill, usually with more or less irritation or inflammation of the mucous membranes of the nose and throat, often associated with catarrhal conjunctivitis and blepharitis (pink-eye). There is always a feeling of lassitude, passing into unaccountable depression, physical and mental, great soreness in the back and limbs, aching of the muscles, more or less stiffness of the neck, cutaneous hyperesthesia, headache, dyspnea and anorexia.

The *temperature* is decidedly irregular; it may rise high; it is often subnormal for more or less protracted periods, and is subject to extreme fluctuations. The *pulse* is not much accelerated; it ordinarily is weak, the heart sharing in the general depression. The *tongue* is coated; the *bowels* are often constipated; the *urine* is scanty and high-colored, or profuse and light-colored. The *skin* is hot and dry and very sensitive to the touch. Sometimes there are irregular perspirations.

In the *catarrhal type*, sneezing, coryza, watering of the eyes, odynphagia, painful respiration and a dry, harassing, irritative cough are early and prominent symptoms. The difficulty and distress in breathing are out of all proportion to the phenomena elicited by auscultation and percussion. Later, the cough becomes freer, and associated with expectoration of a thick, glairy mucus, sometimes blood-streaked or discolored; the nasal discharge becomes thicker and even purulent and hemorrhagic. With recovery, profuse, watery diarrhea occurs. In some cases, symptoms of tonsillitis or of catarrhal pharyngitis are the earliest, those of laryngitis and of bronchitis following. Coryza may be a late manifestation. In some cases, there is a peculiar edematoid condition of the tonsil, uvula and neighboring structures that has been termed "solid edema," as puncture gives exit not to serum, but to a sanious, stringy, lymphoid material. Suppurative otitis media, preceded by excruciating earache and usually affecting both ears in succession, may be the only manifestation in the upper air-tract. This otitis is quite common in children and usually runs through a household.

In the *thoracic type*, pleuritis, pericarditis, endocarditis, pneumonia—catarrhal, croupous or hemorrhagic—or edematous or hemorrhagic infiltration of the lung may cause grave symptoms or even death.

In the *abdominal* or *gastro-intestinal type*, vomiting and purging are common, and there may be great pain and tenderness in the epigastrium or over the entire abdomen. Catarrhal jaundice may occur.

In the *cerebral* or *nervous type*, the headache is intense; there may be insomnia, photophobia, tinnitus aurium, talkativeness or even mild delirium. There is decided hyperesthesia; there may be restlessness, tremor, muscular twitchings or jactitations.

In the *typhoid type*, the course of the disease may be protracted, and the temperature may remain continuously elevated. The prostration may be extreme; the mental phenomena may be characterized by depression. There may be epistaxis, pain in the splenic region, special tenderness in the right iliac fossa and considerable diarrhea. There may be anomalous eruptions —papular, herpetic or erythematous. The action of the heart may be exceedingly feeble. Heart-failure is always a threatening danger.

The *duration* of influenza is as variable as are the symptoms. It may be less than forty-eight hours; it may be several weeks. Commonly, the acute symptoms last from three or four days to a week, but the weakness and depression continue much longer. *Convalescence* may be tardy, prolonged subnormal temperature and annoying perspiration being notable features.

In addition to the *complications* mentioned, there may be hemorrhages from various organs, meningitis—cerebral and spinal—multiple neuritis, arthritis and nephritis. Among the *sequelæ* are tuberculosis, paralyses of various kinds, hemicrania and melancholia, and other psychoses. Of itself influenza is not often fatal or directly provocative of serious complications or sequelæ; but it aggravates existing lesions or morbid processes, reawakens latent disease or searches out the weak point in the organism and renders this liable to the action of exciting causes. Hence, the great variability in its clinical course, and hence, too, the high mortality it occasions among the previously sick and debilitated, among infants and the aged.

With what diseases may influenza be confounded?

The *diagnosis* of influenza may be extremely easy or extremely difficult. According to its type, it may be mistaken for simple catarrhal inflammation of the mucous membrane of the eye, ear, nose, throat, bronchi, stomach or intestines; for measles; for cerebro-spinal meningitis; for acute articular rheumatism; for dengue; for ordinary types of pleurisy or pneumonia; for malarial fever; for acute tuberculosis; for typhoid fever.

Upon what does the discrimination depend?

In times of prevalence of epidemic influenza, or of the analogous epizoöty, the knowledge of that fact will cause one to be on the lookout for the disease, and he may, perhaps, even call other affections by its name. Characteristics upon which stress should be placed are the sudden onset, the great depression, the cutaneous hyperesthesia, the lumbar and muscular pains and the excessive respiratory distress. These serve to distinguish it from indigestion, gastro-intestinal catarrh, bronchitis, coryza and "colds." In contradistinction from typhoid fever or typhus fever, the common occurrence of some form of catarrhal symptoms, the irregularity of the temperature, the shorter duration and the absence of the characteristic symptoms to be described, are additional discriminating points. The differential diagnosis from the other diseases mentioned will be successively developed.

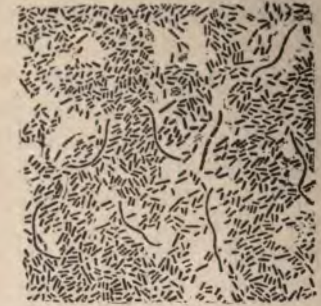
Typhoid Fever—Enteric Fever.

What is typhoid fever?

Typhoid or *enteric fever* is an acute, infectious disease, belonging to the group of essential, continued fevers, dependent upon a specific microörganism, and presenting constant lesions: inflammation, swelling, softening and ulceration of Peyer's patches, enlargement and softening of the mesenteric glands and tumefaction of the spleen. It runs a course of about twenty-four days, beginning gradually and terminating by lysis. The disease is most common in young adults and in the autumn.

The *typhoid-bacillus* (Fig. 1) is a plump, motile organism, with rounded extremities. It is about one-third as long as the diameter of a red blood-corpuscle and one-third as wide as it is long. It presents a characteristic growth upon potatoes and does not liquefy gelatin. It is best stained by means of an alkaline solution of methylene-blue or by carbol-fuchsin or anilin-water fuchsin. It differs from other organisms found in the stools in not generating indol. The bacillus has been found during life in the blood, in the urine and in the stools, and, after death, in the intestinal wall, in the mesenteric glands and in the spleen.

FIG. 1.



Bacilli of typhoid fever. (Von Jaksch.)

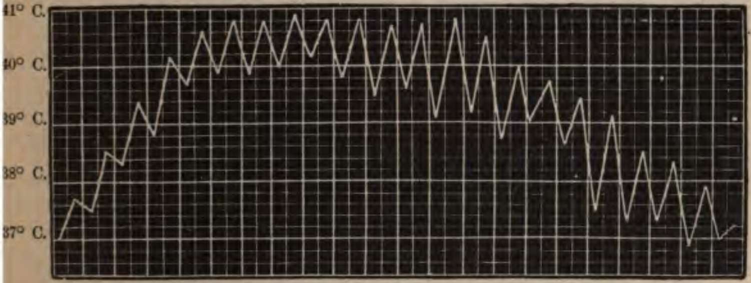
What is the clinical course of typhoid fever?

The *period of incubation* of enteric fever varies between two and eleven days. The *onset* is usually insidious, occasionally abrupt, with a chill. The *prodromal period* is characterized by headache, pains in the back, epistaxis, general malaise, disturbed sleep, loss of appetite and coated tongue. These symptoms become aggravated, while to them are added relaxation of the bowels, abdominal tenderness, especially in the right iliac fossa, intestinal gurgling and elevation of temperature.

The *temperature* pursues a characteristic course. (Fig. 2.) There is a period of gradual ascent (first week), a period of maintained height (second week and half of third week), and a period of gradual fall (last half of third week and fourth week).

For from five to seven days the temperature rises two or three degrees each evening, to recede a degree or a degree and a half on the following morning, until it has reached a level of from 102.5° to 104° F. (in grave cases even higher), at which, with slight evening remissions and morning exacerbations, it lingers for from ten to fourteen days, then to decline inversely pretty much as it rose, until, with the setting in of convalescence, it may fall below the normal.

FIG. 2.



Temperature-chart of a case of typhoid fever. (Wünderlich.)

The *pulse* is accelerated, but not, as a rule, in proportion to the rise of temperature. It may not exceed 90. It is peculiarly soft and rebounding, giving rise to an apparent duplication known as *dicrotism*. The *tongue* is coated in the middle, but red at the margins and tip. The coating is thick and, at first, white. The red, uncoated portion at the tip occupies a characteristic wedge-shaped area. The lips, gums and teeth become covered with sordes. The patient often exhales a characteristic odor. The *pupils* are dilated. A limited flush colors the cheek.

Toward the end of the first week or early in the second, a varying number of small, slightly elevated, *rose-colored spots* that disappear upon pressure or upon stretching the skin may be observed upon the trunk—upon the abdomen or chest, anteriorly or posteriorly. These spots appear in successive crops, each of which lasts for several days.

The *urine* presents a characteristic reaction. If to one part of a (one-half per cent.) solution of sodium nitrite in distilled water (1 : 40) and forty parts of a saturated solution of sulphanilic acid in dilute hydrochloric acid (1 : 20) is added an equal bulk of urine and the whole is rendered alkaline with ammonia-water, a deep-red color is produced.

In classically typical cases, there is *diarrhea;* but in almost an equal number, however, the bowels are constipated. The diarrheal stools present a characteristic appearance, being thin and yellowish, "ochrey," or like pea-soup; they have a peculiar, fetid odor; sometimes they contain blood, independently of a formal hemorrhage. Tympanites is common. The area of *splenic percussion-dulness* is *increased.* In the second week, if not earlier, *mental dulness* and listlessness are manifest. The patient pays little heed to his surroundings, but usually responds when spoken to. Deafness and visual disturbances are not uncommon, and there is frequently low delirium. In the third week, the first sound of the heart is observed to be feeble; *emaciation* is decided, and the *tongue* often becomes dry, fissured and coated with a heavy, brown fur. Intestinal hemorrhage may occur. Perforation of the bowel, with consecutive peritonitis, is among the dangers.

Usually, towards the end of the third or the beginning of the fourth week, progressive improvement is manifested, coincidently with the decline of temperature. The morning remissions exceed the evening rises. Commonly by the twenty-fourth day, but often much later, the temperature has fallen to the norm. Convalescence is slow.

Death may take place in or after the second week, from exhausstion, toxemia, fever or the accidents of the disease.

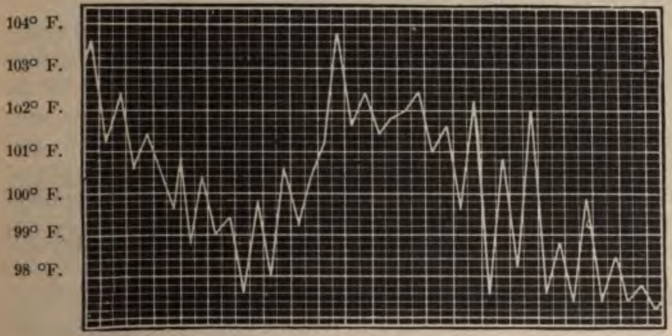
Other *complications* and *sequelæ* than those mentioned are bedsores, phlebitis, thrombosis, endocarditis, parotiditis, edema, inflammation or ulceration of the larynx, bronchitis, pleuritis, pneumonia, pulmonary tuberculosis, osteomyelitis, meningitis, peripheral neuritis, the formation of abscesses and gangrene.

Sometimes the symptoms are mild, constituting *ambulatory* or *walking typhoid fever.* At other times, the attack, while perhaps severe, terminates at the end of a week, or of two weeks, constituting *abortive typhoid fever.* A few days after convalescence has set in, the temperature may again rise, and the attack be repeated, though curtailed in duration, constituting a *relapse*. A relapse may likewise interrupt the declining course of the disease. Sometimes, when convalescence is apparently about to set in, the temperature reascends and remains elevated for an uncertain period, constituting a *recrudescence*.

What is the distinction between a relapse and a recrudescence in typhoid fever?

A typical *relapse* in typhoid fever includes a redevelopment of the entire group of morbid phenomena of the primary disease, as indicated by a characteristic temperature-curve, splenic enlargement and rose-rash, though the duration may be shortened.

Fig. 3.



Temperature-chart of relapse in typhoid fever. Convalescence from the relapse interrupted by recrudescence. (From a case at the Philadelphia Hospital.)

A *recrudescence* refers only to a reappearance of fever. The temperature of recrudescence does not pursue a typical course; it may fall as suddenly as it rose. A recrudescence may depend upon some complication or some accidental source of irritation, *e. g.*, peritonitis, or constipation or the premature or injudicious taking of food. A relapse indicates renewed activity of the specific cause of the disease.

How is perforation of the bowel in typhoid fever to be recognized?

Perforation of the bowel in the course of typhoid fever may or may not be preceded by intestinal hemorrhage. When it occurs, the patient experiences a sudden and intense pain, localized at one spot in the abdomen, but soon extending. There may also be rigors. Tympanites, if absent, develops; if present, it increases. Vomiting may occur. There is exquisite abdominal tenderness; the patient lies upon his back, with the legs drawn up; the *face* is pale, pinched and anxious; the *pulse* is small, hard and rapid; the *breathing* is shallow and thoracic. *Shock* and *collapse* are common. With the fall of temperature, the patient's mind may become clear. *Death* may take place in a few hours; but more commonly the temperature again rises and the symptoms of *peritonitis* become predominant, death occurring in the course of a few days.

Fatal perforation may, however, occur without decided symptoms either of shock or of peritonitis; or there may be a sudden fall or a sudden rise of temperature, a sudden increase in the pulse-rate or in the intensity of the prostration, or there may be sudden vomiting; or there may be simply persistent and rebellious tympanites, with comparatively slight abdominal tenderness and, perhaps, marked depression in the general state of the patient. While perforation is usually fatal, recovery has occurred in well-authenticated instances.

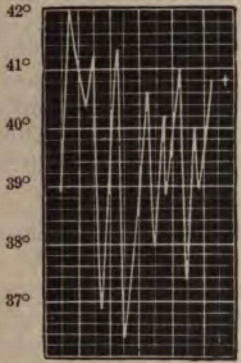
How are typhoid fever and pyemia to be differentiated?

Pyemia may be attended with typhoid symptoms, diarrhea and cerebral manifestations. The temperature (Fig. 4), however, pursues a different course from that of typhoid fever; it is irregular and presents wide variations in range, often declining below the normal. The morning temperature may be normal, that of noon several degrees above normal, that of evening lower than that of noon. There may be great and sudden changes from day to day. In addition, there may be recurrent chills and sweats. The rose-rash is wanting. The detection of a primary focus of suppuration and the results of metastasis point to the cause of the equivocal symptoms.

How are typhoid fever and a typhoid condition to be differentiated?

A condition of asthenia and low vitality developing in the course of certain febrile conditions, surgical affections, septicemia and pyemia, possibly attended with diarrhea, is to be distinguished from typhoid fever by the previous history: on the one hand, there are the evidences of some constitutional or local disease; on the other, epistaxis, headache, continuous fever of gradual invasion. The age of the patient may have weight: typhoid fever being infrequent after thirty-five and rare after fifty, while in the aged many diseases, especially pneumonia, commonly assume a typhoid type. A rose-rash is significant of typhoid fever.

FIG. 4.



Temperature-chart of a case of pyemia. (Wunderlich.)

How are typhoid fever and yellow fever to be differentiated?

Initial headache and pains in the loins attend both typhoid fever and yellow fever; but, in the latter, epistaxis and diarrhea are wanting, the onset is abrupt and the symptoms remit on the second or third day. The discoloration of the skin that gives its name to yellow fever is wanting in typhoid. Yellow fever is a disease of hot climates; typhoid fever, one rather of temperate climates.

How are typhoid fever and variola to be differentiated?

For several days it may be impossible to distinguish typhoid fever and variola from one another. Both present headache and pains in the back. Epistaxis and diarrhea may be wanting in typhoid fever. The onset of variola, however, is likely to be abrupt; that of typhoid fever insidious and gradual. On the third or fourth day, there appears in variola a characteristic eruption; simultaneously, the temperature declines. The eruption of typhoid fever is unlike that of variola and rarely appears before the fifth day. The subsequent course of the two diseases is sufficiently diverse to remove any possibility of confusion. The presence or absence of vaccine protection and the existence of other cases of one or of the other disease may have some weight in the early diagnosis.

How may one avoid confounding pneumonia with typhoid fever?

While pneumonia may present symptoms of a typhoid character and a temperature-course not unlike that of typhoid fever, the respiratory frequency is out of all proportion to the pulse-rate, while careful physical examination will reveal the signs of pulmonary consolidation (dulness on percussion, bronchial breathing, crepitant râles, increased vocal fremitus and vocal resonance) and perhaps the friction-sound of an associated pleurisy. Pleurisy, however, is by no means rare early in the course of typhoid fever. The appearance of rusty sputum clinches the diagnosis of pneumonia. It must, however, be borne in mind that pneumonia may occur as a complication of typhoid fever. In such a case, the association must be recognized by the rose-rash, the splenic enlargement, the diarrhea and the protracted course of the disease.

How are typhoid fever and trichiniasis to be differentiated?

Trichiniasis may present many of the manifestations of typhoid fever, but the epistaxis, the severe headache, the enlargement of the spleen, the characteristic stools, the typical temperature-curve and the rose-spots are usually wanting. The knowledge that there is such a condition as trichiniasis, with inquiry as to the food taken and the detection of nodules in the painful muscles, ought to be sufficient to prevent mistake.

Typhus Fever.

What are the symptoms of typhus fever?

Typhus fever, also called *famine-fever*, *ship-fever* and *jail-fever*, is an acute, infectious, essential fever, a type of contagious disease. It develops in crowded and unwholesome places, among the poor and wretched, but it may be communicated to any. It has a *period of incubation* of variable duration—from a few hours to two weeks, during which the patient is comparatively comfortable, or there may be a brief stage of preliminary depression.

With the *onset* of the disease there are general malaise, headache, perhaps a chill, pains in the back and a heavily coated tongue, perhaps with nausea. The *temperature* rises to between 104° and 106° F.; the *pulse* is frequent, at first full, but early becoming feeble. Stupor soon develops. The *bowels* are usually constipated. The *expression* is dull. The *conjunctivæ* are injected. The *pupils* are usually contracted. The *face* appears livid. A *musty odor* is manifest. The *body* may be covered by a diffuse, red rash. Between the fourth and the sixth day a *coarse, papular eruption* appears, usually on the trunk and extremities, exceptionally on the face. Intermingled with this are many petechial spots. For two or three days new papules appear, to recede gradually and disappear.

At the close of the first week or early in the second, low, muttering delirium, or coma-vigil, without great restlessness or with ceaseless tossing, muscular twitching and jactitation, appears; the mental depression is profound. In the *cerebral type* there is a wild, fighting delirium, with intolerance of light and illusions of sight and hearing. Excitement is soon succeeded by weakness and prostration, perhaps by fatal coma. In some cases the *respiration* is shallow, irregular and noisy, though no change in the lungs can be detected. The *heart-sounds* are feeble, though the beat may be excited. Often, there develops an endocardial murmur, due to the depraved state of the blood. The *tongue* is brown and cracked, the teeth and gums covered with sordes. The *urine* is scanty, high-colored, deficient in chlorides and may contain albumin.

In cases that recover the temperature gradually subsides at the end of the second week, a decided decline taking place on the fourteenth or sixteenth day, accompanied, perhaps, with profuse perspiration, diarrhea or a copious discharge of urine. *Relapses* are rare. An attack confers subsequent *immunity*. Pulmonary *complications* are the most common. Others may be meningitis, phlebitis, gangrene, erysipelas, parotiditis, edema of the larynx. During the last stages, or after convalescence, acute tuberculosis may develop.

How are typhoid fever and typhus fever to be differentiated?

Typhus fever is contagious; typhoid is not. Typhus is the more likely to be epidemic. Prodromata are more common and of longer duration in typhoid. The onset is acute in typhus, insidious in typhoid. Typhus lasts about two weeks; typhoid not less than three. The eruption of typhoid consists of small rose-spots, usually confined to the abdomen and chest, and appearing in successive crops; that of typhus is coarse, macular and petechial and of more extensive distribution. The skin is usually moist in typhoid; it is dry in typhus. In typhus, the body exhales a characteristic musty odor. The bowels are often loose in typhoid fever; they are usually constipated in typhus. Nervous prostration is the more profound in typhus. The course of the temperature is different in each. Epistaxis is common in typhoid; uncommon in typhus. In typhoid fever, one finds on post-mortem examination intestinal ulceration and enlargement of the spleen and of the mesenteric glands; in typhus, no constant lesions; though the spleen is likely to be diffluent. Typhoid fever and typhus fever may, though rarely, coëxist in the same patient.

How are variola and typhus fever to be differentiated?

Both variola and typhus fever are in a high degree contagious. Should both be simultaneously epidemic, the diagnosis may be difficult during the first few days of the attack. The eruption of variola, however, appears from twenty-four to thirty-six hours earlier than that of typhus. The former is usually situated on the face, as well as on the trunk, and passes successively through the stages of papule, vesicle and pustule, the pustules rupturing and leaving cicatrices. The eruptionof typhus fever rarely or never appears on the face ; it remains largely papular, though in part it becomes petechial. In typhus fever, the temperature becomes high at the onset and continues high ; in variola, the temperature declines with the appearance of the eruption. Vaccination commonly protects against variola ; it affords no protection from typhus fever. Finally, typhus is a disease of about two weeks' duration ; variola, of quite three.

Cerebro-spinal Fever—Epidemic Cerebro-spinal Meningitis.

What are the clinical features of cerebro-spinal fever?

Cerebro-spinal fever or *epidemic cerebro-spinal meningitis*—a specific, essential fever of continued type, associated with a constant lesion—is an acute and extremely fatal infectious disease, varying much in its clinical manifestations, but usually characterized by decided disturbances of the cerebro-spinal functions. In some cases cerebral, in others spinal symptoms predominate. Not infrequently, respiratory phenomena or blood-changes assume great prominence. A characteristic eruption is usually a marked feature.

The attack may develop gradually, but more often it *sets in* suddenly, with a rigor followed by fever ; malaise ; nausea ; great thirst and vomiting, often with a clean tongue and no gastric derangement ; vertigo ; excruciating headache, remitting, but never entirely ceasing, and attended with paroxysmal exacerbations ; rigidity of the head and neck, sometimes passing into opisthotonos ; muscular twitchings or convulsions ; dryness of the skin, with hyperesthesia and paresthesia. There may also be photophobia and tinnitus aurium. Prostration soon becomes profound, though restlessness may continue. Delirium may set in and be followed by stupor and coma. The *expression* is anxious. The *pulse* is rapid and extremely irregular. The *temperature* fluctuates between wide limits. Hyperpyrexia is not rare. It may develop suddenly and persist until death. The temperature may continue to rise after death. A sudden fall of temperature may usher in collapse and death. A gradual fall of temperature precedes recovery. The action of the sphincters is often deranged, so that there may be incontinence of urine or feces, or retention of urine, or constipation. As a rule, retention is an early, incontinence a late symptom. The *urine* is often albuminous and contains an excess of urates.

Between the first and the third day, purpuric spots, or an erythematous eruption that quickly becomes petechial, may appear upon the trunk and extremities. The disease is sometimes called "spotted fever," from the character of this eruption. Between the third and the sixth day, herpetic vesicles may appear on the face about the lips.

In the further progress of the case, the *pupils*, at first contracted, become dilated; paralysis and anesthesia of irregular distribution appear; disturbances of sight and hearing, perhaps also blindness and deafness develop. The *respiration* may be profoundly disturbed. As death approaches, the breathing may assume the Cheyne-Stokes type. Short remissions in the general severity or in individual symptoms may occur, to be followed by renewed exacerbations. The *duration* of the disease is variable. The *fastigium* is commonly reached on the sixth day. In protracted cases, profound emaciation occurs. *Death* may take place early or late, in coma, by exhaustion, or by apnea. If recovery ensues, *convalescence* is tardy, and sometimes protracted, while permanent loss of special senses is common. Pneumonia is a common *complication*. Palsies, headache and epileptiform convulsions may be additional *sequelæ*.

In addition to the ordinary type of epidemic cerebro-spinal meningitis, there may be *fulminant* cases (death occurring within twelve hours), *mild* or *abortive* cases and *protracted* or *typhoid* cases. *Sporadic* cases are rare.

Instances of contagion (direct transference from the sick to the well) and of portagion (conveyance by the person or belongings of those that have been in contact with the sick) of cerebro-spinal fever appear to have been authenticated, but are extremely uncommon.

How are cerebro-spinal fever and tetanus to be differentiated?

Cerebro-spinal fever appears in epidemics, while tetanus usually occurs sporadically, as a result of the infection of a wound by soil. Trismus, an early symptom of tetanus, is the less common in cerebro-spinal fever. Opisthotonos, general rigidity and spasm are more marked in tetanus than in cerebro-spinal fever. Recovery from tetanus is exceptional. Death is not the invariable termination of cerebro-spinal fever.

Tetanus is wanting in the palsies, the eruption, the derangement of intellection and sensation and the febrile symptoms of cerebro-spinal fever, though towards the fatal termination the temperature may rise inordinately high.

How are cerebro-spinal fever and typhus fever to be differentiated?

While both cerebro-spinal fever and typhus fever occur in epidemics, and both may be sudden in onset and attended with profound nervous phenomena and petechial eruption, cerebro-spinal fever has not the dusky, stupid facies of typhus, while the herpes of the face, the retraction of the head, the fixed spinal pain, the muscular rigidity and the heightened sensibility of cerebro-spinal fever are not observed in typhus; nor is typhus, as a rule, accompanied with the great impairment of special senses or followed by the paralytic sequelæ of cerebro-spinal fever. The general course of the two diseases, the fever and the eruption may be discriminated on careful observation. The greatest difficulty occurs in cases of malignant cerebral typhus.

How are cerebro-spinal fever and torticollis to be differentiated?

The muscular contraction that gives rise to torticollis is usually unilateral and limited, while in cerebro-spinal fever the contraction is symmetrical and not confined to the muscles of the head and neck. The symptoms of an acute, febrile disease, with disturbances of the sensorium and paralytic concomitants and sequelæ, are not present in torticollis, but are characteristic of cerebro-spinal fever. Even mild cases of cerebro-spinal fever, lacking the characteristic febrile course and without eruption, will present severe headache.

What are the distinctions between cerebro-spinal fever and smallpox?

Headache, vertigo, nausea, vomiting, pain in the back and fever attend both cerebro-spinal fever and smallpox; but retraction of the head, muscular rigidity and paralysis, hyperesthesia and anesthesia are wanting in smallpox, and the peculiar temperature-record and the characteristic eruption of smallpox are not seen in cerebro-spinal fever.

What are the distinctions between cerebro-spinal fever and yellow fever?

Yellow fever is especially a disease of hot climates; when found elsewhere its importation may be traced. If cerebro-spinal fever display any susceptibility to climatic conditions, it is most common in temperate zones. Characteristic symptoms of motor and sensory derangement, observed in the course of cerebro-spinal fever, are wanting in yellow fever, which is a disease of brief duration, in contrast to cerebro-spinal fever, the duration of which may be protracted. Petechial and herpetic eruptions appear during the progress of cerebro-spinal fever, while yellow fever is characterized by a saffron-yellow color of the skin. The black vomit often seen in yellow fever is entirely wanting in cerebro-spinal fever. Although remissions in the intensity of special symptoms may occur in the course of cerebro-spinal fever, the characteristic "lull" of yellow fever is absent.

How are cerebro-spinal fever and typhoid fever to be differentiated?

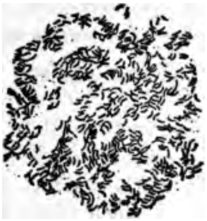
In cerebro-spinal fever, the onset is usually abrupt; in typhoid it is insidious. In typhoid fever the temperature pursues a typical course; in cerebro-spinal fever there is no regularity. The eruption of cerebro-spinal fever is petechial or herpetic and appears early—before the fourth day; that of typhoid is roseolous and appears not earlier than the fifth or sixth day. Constipation is the rule in cerebro-spinal fever; diarrhea often attends typhoid. Nausea and vomiting occur in cerebro-spinal fever, but not usually in typhoid. The retraction of the head, the paresthesiæ and the paralyses of cerebro-spinal fever are all wanting in typhoid fever. The headache is more intense in cerebro-spinal than in typhoid fever; in the latter it disappears when delirium sets in; in the former, delirium and headache coëxist. The knowledge of an epidemic assists in the diagnosis. The discovery ophthalmoscopically of recent papillitis (choked disc) or optic neuritis would be diagnostic of cerebro-spinal meningitis in contradistinction from uncomplicated typhoid fever. In rare cases, typhoid fever is complicated by meningitis.

Asiatic Cholera.

What are the symptoms of Asiatic cholera?

Asiatic cholera, also called *cholera infectiosa*, is an acute, infectious disease, having its home in tropical climates and occurring in epidemics. It is dependent upon a specific organism, which is found in the alvine discharges of the patient and is principally transmitted by means of milk and drinking-water.

FIG. 5.



Comma-bacillus of cholera. (Vierordt.)

The disease has a *period of incubation* of from half a day to three or four days. It may *set in* suddenly, with a chill, but more usually the attack proper is preceded by a moderate diarrhea, to which the name *cholerine* has been given. This constitutes the *first* or *premonitory stage.* The course of the disease may be arrested at this stage.

In the *second stage* (*stage of spasm*, or *stage of serous diarrhea*), there occur violent cramps in the abdomen and legs, and the intestinal flux increases in severity. There is often obstinate vomiting. The patient complains of thirst, is restless and anxious; prostration is marked; the pulse is weak and thready; the skin is cold and shrunken; the eyeballs are sunken. The *temperature*, taken in the rectum or with a thermometer carefully applied and allowed to remain at least ten minutes in the axilla, will be found to be elevated. The *stools* are almost liquid and colorless and contain large quantities of epithelium, constituting the so-called "rice-water" discharges, in which the *comma-bacillus* of Koch (Fig. 5) is to be found.

Soon, a *third* or *algid stage*, or *stage of collapse*, sets in. The *circulation* fails, and there is marked depression of the vital powers; the *respiration* is shallow and accelerated; the *skin* becomes as cold as marble; the *breath* may be chilling; the *voice* is lost. Suppression of urine often occurs. The *urine* that is secreted is albuminous and contains casts.

In this condition of collapse the patient may die, or he may enter upon a *fourth stage*, or *stage of reaction*, convalescence setting in or a low, typhoid condition developing, with fever and delirium and possibly with suppression of urine. This stage may terminate in death or in convalescence. Convalescence may be *complicated* by ulceration of the cornea and by parotiditis.

How are cholera nostras and cholera Asiatica to be differentiated?

Cholera nostras occurs endemically; cholera Asiatica, epidemically, and, in Europe and America, by importation. Asiatic cholera is by far the graver affection; the stools present a characteristic "rice-water" appearance, in which a specific bacillus is to be found. If an apparently similar bacillus be found in the stools of cholera nostras, its morphology and culture will prove it to be different.

How is arsenical poisoning to be distinguished from cholera?

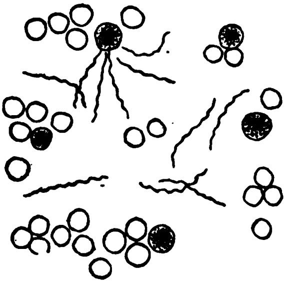
Poisoning by arsenic occasions vomiting, cramps in the abdomen and legs, and diarrhea with stools of a "rice-water" character. Local evidences of the ingestion of arsenic may be present in the mouth; vomiting precedes diarrhea; the stools are bloody and do not contain the specific comma-bacilli.

Relapsing Fever.

What are the distinguishing features of relapsing fever?

Relapsing fever is rare in other than certain tropical countries. It is a specific, essential fever, of intermittent type, and is dependent upon the presence of a specific organism—the *spirocheta Obermeierii*—in the blood. (Fig. 6.)

FIG. 6.



Spirochetæ of relapsing fever. (V. Jaksch.)

The growth and development of the parasite give rise to the periodic paroxysms that characterize the disease and give it its name. After a *period of incubation*, varying from several hours to two weeks, the disease *sets in* suddenly with a chill, followed by fever, with decided elevation of *temperature*, muscular pains, vertigo, headache, nausea and vomiting. The spleen is enlarged. Often there is jaundice.

In the course of from five to seven days the attack abates with the suddenness with which it set in, the temperature falling to the normal and profuse diaphoresis occurring.

For about a week the patient is free from symptoms. At the end of this time, the paroxysm is repeated, to again subside; and this may happen a number of times.

Convalescence is tardy and protracted. During the paroxysms, spirochetæ in large numbers may be found in the blood. They are not to be found during the intermissions.

How is relapsing fever to be diagnosticated from typhoid fever?

In typhoid fever, both onset and subsidence are gradual; in relapsing fever, they are sudden. The former is a continued fever; the latter intermittent and periodic. Relapsing fever does not present the rose-rash of typhoid fever. Jaundice, often present in relapsing fever, does not occur in typhoid fever; nor are spirochetæ found in the blood in typhoid fever.

How does relapsing fever differ from typhus fever?

The course of relapsing fever is interrupted by intermissions, giving rise to a disease of periodic type; typhus is a continued fever.

The marked nervous symptoms, as well as the exanthem, of typhus are wanting in relapsing fever.

Spirochetæ are found in the blood only in relapsing fever.

Upon what does the differential diagnosis between relapsing fever and yellow fever depend?

Yellow fever is a disease of not more than three stages; there may be more or less in relapsing fever. The primary acute stage, as well as the period of intermission or remission, is longer in relapsing fever than in yellow fever. Vomiting occurs late in yellow fever, early, if at all, in relapsing fever. Vertigo is a more marked symptom in relapsing than in yellow fever. The occurrence of one or the other in an epidemic might afford a clue in diagnosis. Spirochetæ are present in the blood in relapsing fever; they are not found in yellow fever.

Malarial Diseases.

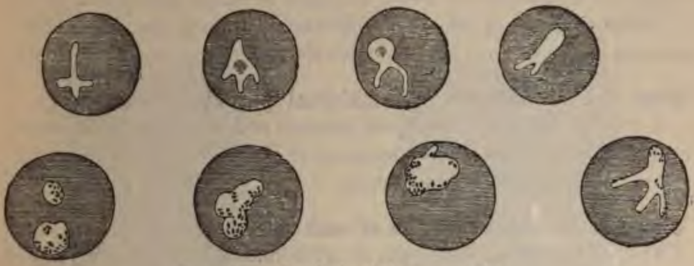
What are the characteristics of malarial disease?

The group of diseases known as *malarial* is characterized by paroxysmal periodicity, enlargement of the spleen and liver, melanemia and the presence in the blood, free or within the red corpuscles, of parasites that exert a deleterious influence upon the red cells. (Fig. 7.)

During the paroxysm, the urine is said to be irritant, the proportion of both water and solids being increased.

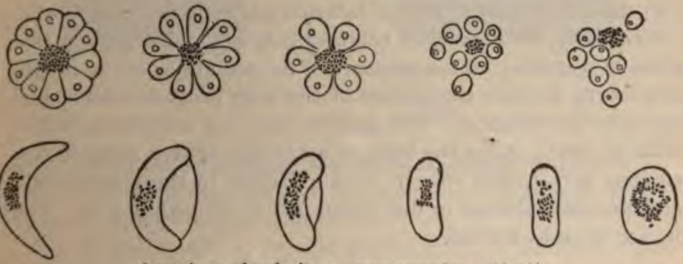
Malarial diseases prevail principally in the lowlands of warm climates, with marshy soils.

FIG. 7.



Organisms of malaria—intracorpuscular.

FIG. 8.



Organisms of malaria—extracorpuscular. (Macé.)

What are the varieties of malarial fever?

Malarial fevers are said to be *intermittent* when between two paroxysms there intervenes a period of freedom from symptoms, with a restoration of the temperature to the normal; *remittent*, when between two paroxysms the symptoms moderate and the temperature falls (but not to the normal level).

When the paroxysm is repeated daily, the fever is designated *quotidian;* if repeated on alternate days, *tertian;* if with an interval of two days, *quartan.* If two paroxysms occur daily, the fever is called a *duplicated quotidian.* There may be a *double tertian*, in which occur daily paroxysms, of which only those of alternate days are alike; a *double quartan*, and other combinations. When the paroxysms succeed one another so closely that the cold stage of one begins before the sweating stage of its predecessor ends, the fever is called *subintrant*.

Morphologic and biologic differences among the organisms present in the various types of malarial fever have been recorded.

What are the features of a malarial paroxysm?

A typical malarial paroxysm consists of a *cold stage*, a *hot stage* and a *sweating stage*. The disease is therefore called "chills and fever." It is also known as *ague*.

What are the characteristics of each stage?

The *cold stage* or *chill* sets in with malaise, nausea, vertigo, shivering; as the *rigor* becomes more pronounced, the patient may be severely shaken; his teeth chatter; the *skin* is cold and rough; the *breathing* is shallow and hurried; the *pulse* is small and rapid; the *temperature* of internal parts, however, is febrile.

Gradually, the feeling of coldness subsides and gives way to a sense of warmth, the temperature of the rectum or of the mouth continuing to rise; the surface of the body becomes flushed and the eyes are brilliant. The patient has now subjective sensations of *fever*. After the lapse of a variable time, a more or less copious *perspiration* sets in, with a decline in the temperature and an amelioration or disappearance of the symptoms. The paroxysm is at an end.

What are the clinical features of malarial intermittent fever?

In *intermittent fever*, as the name indicates, there is in the interval between two paroxysms a complete intermission of the symptoms, the temperature becoming normal or subnormal.

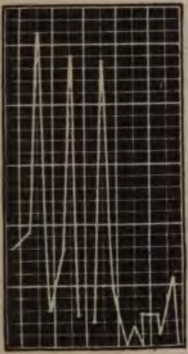
The *cold stage* lasts from fifteen minutes to an hour, the *hot stage* and the *sweating stage*, respectively, a varying number of hours. The beginning of each successive paroxysm anticipates in time of the clock that of the preceding paroxysm.

Untreated, the paroxysms of intermittent fever lose their regularity.

The disease may gradually and spontaneously subside, or the paroxysms may become remittent or pass into the malarial cachexia.

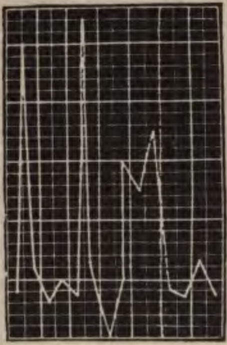
After apparent recovery, a tendency to a return of the paroxysms on the fifth, seventh, ninth or fourteenth day is sometimes observed.

FIG. 9.



Temperature-chart of quotidian intermittent fever. (Wunderlich.)

FIG. 10.



Temperature-chart of tertian intermittent fever. (Wunderlich.)

What are the clinical features of malarial remittent fever?

Remittent fever represents a more profound degree of intoxication than does intermittent fever.

The *chill* is usually severe and protracted. In addition, there is gastric irritability, perhaps vomiting, sometimes jaundice. The *temperature* attains a high degree. The *hot stage* may last for from six to eighteen hours and is followed by profuse *perspiration*.

In the *interval* between two paroxysms, the symptoms moderate and the temperature declines, but does not reach the normal.

After the occurrence of several paroxysms, the chill may be wanting; or there may be but one—the initial chill. Without medicinal intervention, the remissions may gradually become less decided and a typhoid condition may develop.

What are the symptoms of hemorrhagic malarial fever?

Hemorrhagic malarial fever is a grave form of malarial intoxication in which the height of the paroxysm is marked by headache, severe pain in the back, nausea, vomiting, decided jaundice and hemorrhages from various mucous surfaces, particularly from the kidneys.

What is pernicious malarial fever?

In certain localities in which the malarial organisms are exceedingly numerous or virulent, the attack manifests a *pernicious* tendency. The clinical picture depends upon the system that bears the brunt of the disease. There may thus be a *cerebral form*, characterized either by delirium and excitement or by coma and depression; or there may be a *thoracic form*, in which the respiration is accelerated, and there is an urgent sense of the need of air; or there may be a *gastro-intestinal variety*, attended with nausea, vomiting, jaundice and diarrhea; or there may be an *asthenic* or an *algid variety*, in which there is a condition of marked debility and a striking coldness of the surface and of the breath. Pernicious malarial fever usually manifests its character only after a preliminary paroxysm of apparently ordinary intermittent or remittent fever. Unless promptly and vigorously treated, it is likely to be fatal.

What are the symptoms of the malarial cachexia?

After protracted residence in a malarious district or following untreated or rebellious intermittent or remittent fever, there may occur irregular chilly sensations, with some tendency to periodicity, an occasional sense of feverishness, headache, mental torpor, drowsiness, a sallow complexion, constipation or diarrhea, impaired appetite, enlargement of the spleen and liver—a complex of symptoms that may not yield to medicinal treatment, but which improves on removal to a non-malarious climate.

What is meant by "Dumb Ague?"

There is a variety of irregular manifestations of malarial or paludal poisoning, sometimes acute, but more frequently subacute or chronic, which do not at any time present the classical picture of chill, fever and sweat. These attacks of "dumb ague," "masked malaria," "larval paludism," as they are variously called, comprise chilly sensations, irregular fever, or flushes, or subjective sensations of heat, joint-pains and muscle-pains, headache and neuralgias of various kinds, cough, with or without bronchial or laryngeal signs, gastric and other visceral disturbances, sometimes taking the form of crises; in severe or protracted cases, anemia, anasarca and albuminuria, hematuria and hemoglobinuria have been observed. A common variety is the so-called "brow-ague," a form of frontal headache frequently associated with tenderness of the nerves at the supra-orbital and infra-orbital foramina and sometimes with an intensely painful sensation of pressure or constriction referred to the nasal bones; the manifestations usually exhibiting an irregular periodicity. Appearing in the morning and disappearing at night, or when aggravated by sunlight, it is called "sun-pain."

Enlargement of the spleen is sometimes demonstrable. A careful study of the phenomena in these cases will usually elicit some periodicity in their recurrence, or while the prominent symptoms, gastric or neuralgic, or whatever they may be, may not be periodic, careful temperature-observations will show an unsuspected periodic rise. Sometimes, if quinine be administered for a short time, and then withheld, a distinct periodicity in the symptoms will be developed upon withdrawal of the drug. The discovery of characteristic plasmodia in the blood would establish the diagnosis. The organisms are usually comparatively few in number and of the *crescentic* or *sickle-shaped* variety.

What is meant by Ague-cake?

The enlargement of the spleen in a case of chronic malaria or of malarial cachexia is sometimes quite manifest to ordinary inspection. When not so readily manifest to sight, it may easily be detected by palpation. Its connection with malaria being well known, the enlarged spleen has in vulgar parlance acquired the name of *ague-cake.*

What are the distinctions between malarial fever and the fever of suppuration or of septic infection?

Suppuration and septic infection usually give rise to fever of a remittent or intermittent type. When an obvious cause exists, the recognition of the nature of the fever is easy. When, however, the fever is induced by deep-seated suppuration, as when abscesses form or in case of pulmonary tuberculosis or of occlusion of the hepatic or of the common bile-duct, the connection may be obscure. The distinguishing features, however, are that the symptoms of suppurative or of septic fever are rarely of regular periodicity or typical in course; that they are often uninfluenced by the administration of quinine, which acts specifically in the malarial diseases; and that the hematozoa characteristic of malarial fever are wanting.

With what conditions may pernicious malarial fever be confounded?

The *gastro-intestinal* type of the disease may simulate ordinary gastro-intestinal catarrh, but the one is a grave condition, while the other is not; the one is febrile the other antifebrile; the one submits to treatment by quinine, while the other does not; in the one, the blood contains characteristic hematozoa and blood-pigment, while the other does not; in the one, the spleen is enlarged, in the other, it is unchanged in size.

The pulmonary type may be confounded with pleurisy or with pneumonia, but the physical signs and many symptoms will clear up any doubt. In doubtful cases, examination of the blood and treatment with quinine will furnish irrefutable evidence.

The *algid* variety resembles cholera, but it does not occur in epidemics, the general symptoms of cholera are wanting, and treatment decides the result.

Jaundice attends the *hemorrhagic form;* hemorrhages take place from various mucous surfaces and blood is found in the urine. Hemorrhagic malarial fever differs from paroxysmal hemoglobinuria in not being dependent upon exposure to cold, while the urine contains red corpuscles and not merely hemoglobin. Quinine cures the one, but fails to influence the other.

The cerebral type is to be distinguished from those conditions that give rise to apoplexy and from profound intoxications of various kinds. The essential, distinguishing features are the occurrence of the symptoms in the course of an attack of malarial fever, the absence of palsies and localizing symptoms, the tendency to recovery under treatment, the enlargement of the spleen, the presence of hematozoa in the blood.

How are typhoid fever and malarial fever to be differentiated?

Typical cases of intermittent and remittent fever are not likely to be confounded with typhoid fever, but if an intermittent or remittent has existed for some time, uninfluenced by medication, a typhoid condition develops, and the symptoms may occasion some doubt in diagnosis. Under such circumstances, the previous history must be considered.

The diarrhea, the rose-spots, the temperature-course of typhoid fever are all different from what is seen in malarial fevers.

The reaction of the urine to sulphanilic acid and sodium nitrite, described by Ehrlich, and a characteristic bacillus are not found in malaria. The detection of the plasmodia of malaria in the blood dissipates even the remotest doubt.

Malarial fever and typhoid fever may coëxist as so-called typho-malarial fever.

How does syphilitic fever differ from malarial fever?

When secondary syphilis is marked by fever, the elevation of temperature usually occurs at night and is associated with bone-pains, cutaneous eruption and other evidences of syphilis. Cerebral and meningeal syphilis may also give rise to febrile movement. In many cases, the discovery of the plasmodia in the blood and the results of treatment by quinine on the one hand, and the results of treatment by mercury and iodides on the other hand, must make the diagnosis.

Yellow Fever.

What are the characteristics of yellow fever?

Yellow fever is a specific, epidemic disease of hot climates, occurring in a single paroxysm of three stages: the *first*, a *febrile stage*, lasting from thirty-six to forty-eight hours, which sets in with a chill, followed by fever, with capillary congestion, especially of the face and eyes, pains in the head, the back and the calves of the legs, restlessness and anxiety, irritability of the stomach, vomiting, thirst, constipation; the *second*, a *stage of* *remission* or *lull*, of less than six hours, in which the fever subsides and the skin assumes a deep-yellow or bronze hue; the *third*, a *stage of renewal*, in which the symptoms reappear, prostration becomes pronounced and hemorrhages take place from various mucous surfaces; the vomited matters present a characteristic *black* appearance. The urine usually contains albumin and often casts. Suppression of urine may occur.

The *mind* is usually clear almost up to the moment of death, but in some cases delirium and stupor develop.

Death may result from collapse or with convulsions and the symptoms of uremia. If recovery take place, *convalescence* is often gradual, and may occasionally be interrupted by *relapse*. Some cases are quite *mild*, recovery taking place at the end of the first stage. Even grave cases may be so mild in the first stage as to be unrecognized; the patient *walking* about, to be suddenly seized with prostration, quickly followed by *black vomit* and death.

An attack protects against subsequent infection.

What are the distinguishing features between yellow fever and malarial remittent fever with jaundice?

Yellow fever is epidemic; remittent fever, endemic. Yellow fever is a disease of a single paroxysm, not lasting more than a week; remittent fever is a disease of repeated paroxysms, of periodic recurrence, and lasts more than a week.

In yellow fever, the eyes become injected and watery, the expression anxious or fierce. In remittent fever, there is no especial change in the eyes or in the expression.

Prostration and muscular pains are decided in yellow fever and are not so prominent in malarial fever.

Delirium is common in bilious remittent fever, and the mind is always dull. Delirium is not common in yellow fever, and the mind is usually clear.

The pulse may become very slow in yellow fever; it is always quick in remittent fever.

Hemorrhages from mucous surfaces take place in yellow fever; not in ordinary remittent fever.

The urine of yellow fever contains albumin, and suppression may take place; the urine of remittent fever contains no albumin and suppression does not commonly occur. Bile-pigment gradually disappears from the urine of yellow fever and increases in the urine of bilious remittent fever.

An attack of yellow fever confers immunity from subsequent inflexion; one attack of remittent fever predisposes to other attacks. Yellow fever is commonly fatal, remittent fever rarely fatal.

The treatment of yellow fever is uncertain; remittent fever yields to quinine.

Plasmodia malariæ are never found in the blood in yellow fever; they are diagnostic of malarial fever.

How are hemorrhagic malarial fever and yellow fever to be differentiated?

Both hemorrhagic malarial fever and yellow fever occur in hot climates and are attended with jaundice, hematemesis and other hemorrhages.

Yellow fever, however, is epidemic; hemorrhagic malarial fever, endemic. The former consists of but a single paroxysm, of three stages, including a remission; the latter is marked by a series of paroxysms, each followed by a remission.

Black vomit is the more characteristic of yellow fever; hemorrhage from the kidneys, of hemorrhagic malarial fever. Albumin and casts are commonly found in the urine in yellow fever; not in malarial fever. An attack of yellow fever confers immunity from subsequent infection; an attack of malarial fever predisposes to the occurrence of other attacks. The detection of the plasmodia of malaria in the blood establishes the diagnosis.

How is yellow fever to be distinguished from acute yellow atrophy of the liver?

Yellow fever is epidemic; acute yellow atrophy is sporadic. In acute yellow atrophy, the area of hepatic dulness becomes rapidly and decidedly diminished; in yellow fever, there is either enlargement or no demonstrable change.

Yellow fever is, and acute yellow atrophy is not, attended with a distinct remission in the severity of the attack. Yellow fever is sometimes followed by recovery ; acute yellow atrophy is not.

The injection of the eyes, the pains in the back and extremities, found in yellow fever, are wanting in acute yellow atrophy of the liver.

In acute yellow atrophy, leucin and tyrosin are found in the urine, and while cerebral symptoms are more pronounced than in yellow fever, the temperature never rises so high and may even be subnormal.

Weil's Disease.

What are the symptoms of Weil's disease?

Weil's disease, also called *acute infective jaundice*, is an intermittent febrile affection, usually exhibiting two periods of activity separated by an uncertain interval; the first of a little more, the second of a little less, than a week's duration. The disease may *set in* abruptly with nausea and vomiting. The *temperature* at once rises to a considerable height, but falls decidedly on about the night of the fifth day; subsequently declining gradually until the normal level is reached. After an afebrile period of from twenty-four hours to a week, there is a return of fever lasting a few days or a week.

The *attack* is characterized by headache, vertigo, malaise, debility, somnolence, and, sometimes, nocturnal fever and restlessness, hyperesthesia, diarrhea, muscular pains and jaundice. The *pulse* is small and frequent and sometimes dicrotic. The *respiration* is accelerated. The areas of splenic and hepatic percussion-dulness are increased. The *urine* passed is diminished in quantity and contains bile-pigment, bile-acids, albumin and casts. Hemorrhages from various mucous surfaces may take place, epistaxis, hematemesis, hemoptysis and intestinal hemorrhage. Petechial spots may appear in the skin.

The disease has been observed most commonly in summer and in vigorous young men, butchers and soldiers seeming to display a peculiar proclivity. Similar manifestations have followed poisoned wounds. In fatal cases, degeneration of the liver and kidneys and spleen has been found. Parotiditis, pneumonia, iridocyclitis and motor weakness have been *sequelæ*. In cases that recover, the *convalescence* is protracted.

How are Weil's disease and yellow fever to be differentiated?

Weil's disease and yellow fever probably exhibit a closer resemblance in description than in actuality.

Weil's disease shows a special predisposition for young adults, especially butchers and soldiers; yellow fever occurs in epidemics and does not confine itself to any class of individuals. Diarrhea is the rule in Weil's disease; constipation in yellow fever. The initial stage of yellow fever is of shorter duration; it is earlier attended with a remission; the remission is less complete, and both the remission and final stage are shorter than is the case in Weil's disease. Black vomit is not common in Weil's disease; the injection and excitement are less than in yellow fever.

The Exanthemata.

What are the exanthemata?

The term *exanthemata* or *eruptive fevers* is applied to a group of contagious, epidemic diseases, each depending upon a specific infection and having as prominent signs, fever and specifically characteristic eruptions on the skin and often on the visible mucous membranes. As exanthemata are commonly described scarlatina or scarlet fever; measles or morbilli; French measles or Rötheln; smallpox or variola and its modification varioloid; vaccinia—the usual consequence of vaccination; chicken-pox or varicella. Diphtheria and erysipelas might also be included.

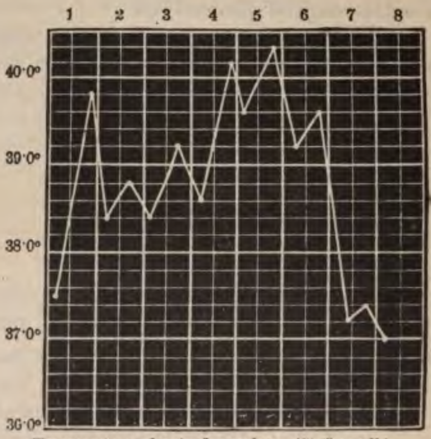
Morbilli—Measles.

Upon what does the diagnosis of morbilli depend?

Morbilli or *measles*, also called *rubeola*, is an acute, contagious disease, common in children, attended with catarrhal symptoms (coryza, rhinitis, pharyngitis, laryngitis, bronchitis, conjunctivitis), febrile elevation of temperature and a characteristic exanthem.

The *period of incubation* is from seven to fourteen days. The *onset* is somewhat abrupt, with a quick rise of temperature to from 102° to 104° F., more or less headache, restlessness, injection and watering of the eyes, sneezing, running from the nose, often swelling of the nose and lip, perhaps cough and slight sore-throat. *Digestion* is commonly disturbed, and the *urine* may be scanty. The *temperature* (Fig. 11) undergoes a considerable elevation with the onset of the attack, declining on the second or third day, to rise again on the fourth, with the appearance of the eruption. This consists of coarse, pink papules, primarily discrete, then becoming surrounded by a somewhat paler border and soon coalescing, to form slightly elevated patches arranged in crescentic form, with intervening healthy skin. The eruption first appears upon the face and neck, then upon the body. The fever declines and the eruption begins to disappear between the fifth and the seventh day. Branny desquamation follows as the attack comes to an end on the ninth day. The eruption may

FIG. 11.



Temperature-chart of measles. (Strümpell.)

usually be detected in the throat a day or two in advance of its appearance on the skin.

An attack of measles commonly protects against subsequent infection; though second attacks are not rare and third attacks not remarkable. The disease is ordinarily mild and benign; sometimes, however, it is malignant and hemorrhagic (*black measles*). Lobular pneumonia and catarrhal otitis media are not uncommon *complications*. An attack of measles is likely to precipitate the development of tuberculosis in one predisposed and to accelerate the course of the disease when it already exists.

How does morbilli differ from typhus fever?

Typhus fever is of longer duration and decidedly more grave than measles, than which it is relatively less common in children. While the eruption of measles in some degree resembles that of typhus, it appears earlier and, as a rule, has no tendency to become petechial; it begins on the face, while in typhus the face escapes. The catarrhal symptoms of measles are wanting in typhus; the profound nervous depression of typhus is not seen in measles. Typhus in North America is rare and as a rule imported, most frequently by emigrant-ships.

Scarlatina—Scarlet Fever.

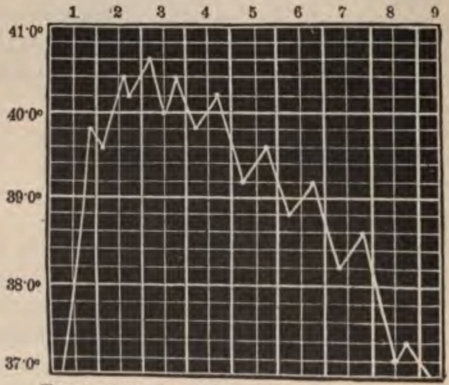
What are the symptoms of scarlatina?

Scarlatina is an acute, contagious disease, to which children and young persons evince a special predisposition. An attack confers relative immunity from subsequent infection.

The *period of incubation* may be short. It varies from twenty-four hours to a week, rarely ten days. The *onset* is usually abrupt, perhaps attended with vomiting or convulsions. The *temperature* at once rises to a considerable height (104° or 105° F.) and the *pulse* attains a striking frequency. In the first twenty-four hours, or sometimes a little later, a diffuse, fine, punctiform, *red rash* appears, at first on the neck and breast and in the flexures of the joints, soon spreading as a uniform scarlet flush over the greater part of the body. Pressure causes temporary dissipation of the redness. There are intense subjective burning and itching of the skin.

The *throat* is usually sore and swallowing is painful. The fauces and palate are reddened, the tonsils and uvula and the adjacent cervical glands are enlarged and there is stiffness of the muscles of the neck. The scarlet discoloration may sometimes be detected in the throat, especially on the free border of the soft palate and on the uvula, in advance of its appearance on the skin. The throat-affection is often ulcerative or pseudomembranous in character. The larynx is rarely invaded. The nose is usually involved and there is more or less serous or seropurulent discharge. The inflammation may extend into the Eustachian tube and involve the auditory apparatus. Suppurative otitis is not an uncommon complication, and perforation and deafness may result.

FIG. 12.



Temperature-chart of scarlatina. (Strümpell.)

The *tongue* is at first heavily coated, but, in the course of a little while, the dense fur is cast off, exposing the surface of the reddened organ, with its enlarged and prominent papillæ—an appearance characteristically described by the designation "strawberry tongue." Thirst is often great. *Digestion* is deranged. The *urine* is scanty.

Severe *nervous symptoms* may occur, twitchings or convulsions, restlessness, insomnia, delirium, stupor, fatal coma; or the nervous disturbances may be very mild and cease with the setting in of convalescence.

In favorable cases, the eruption fades by the fourth or fifth day. The temperature, which has remained high, begins to decline; *defervescence* taking place by somewhat rapid *lysis* (Fig. 12). At the end of a week or nine days, the skin undergoes a furfuraceous or membranaceous *desquamation*, the temperature falls to the normal and convalescence may set in. Persistence of high temperature into the second week is not common and usually denotes a complication. So, too, a sudden recrudescence of pyrexia, after decline has begun, indicates suppuration or other accident. Convalescence may be interrupted by the appearance of symptoms of an *acute nephritis*. Edema of the face and body, with diminished elimination of a dense, high-colored or smoky-looking urine, containing considerable albumin and blood, as well as blood-casts and epithelial casts of the uriniferous tubules. Not rarely, albuminuria and other evidences of renal congestion or inflammation may be detected prior to the appearance of edema. With or without suppression of urine, there may occur convulsions, delirium, stupor, coma and death.

Another rather frequent *complication* is arthritis, which may involve a single joint or several joints. Sometimes, with or without joint-symptoms, there is endocarditis, pericarditis or pleuritis. Permanent valvular lesions of the heart may be *sequelæ*.

Various *types* of scarlatina are observed in addition to the ordinary form. When throat-symptoms predominate, the attack is called "anginose." Sometimes the disease is *mild* or *abortive*. At other times it is *malignant* in virulence. In malignant cases, the rash may be delayed; it may be pale and indistinct or dark and livid. Cases in which the rash is wanting may be mild or severe. This form is called "larval" or "scarlatina sine exanthemate." In these cases, dropsy or suppression of urine may be the first symptom to attract attention. Anasarca may occur without nephritis, but its dissociated occurrence is not common. Scarlatina is said occasionally to be *hemorrhagic*.

How are scarlatina and measles to be differentiated?

Scarlatina usually sets in with vomiting or convulsions; measles rarely so begins. Rather severe sore-throat and glandular enlargement characterize scarlatina; catarrhal symptoms are present in measles. The great rapidity of pulse and elevation of temperature commonly observed in scarlatina are wanting in measles The eruption of scarlatina appears on the first or second day of the disease and is finely punctate, occasioning an appearance of diffuse redness; the eruption of measles appears not before the third day and is commonly papular, arranged crescentically, with areas of intervening healthy skin. After a preliminary elevation, the temperature of measles falls on the second or third day, to rise again with the appearance of the eruption, then to subside rapidly; the temperature of scarlatina at once mounts high and, after a few days, declines gradually. Nervous symptoms are decided in scarlatina, wanting in measles.

Rötheln.

What are the symptoms of Rötheln?

Rötheln, also called *roseola*, *German measles*, *French measles* and, incorrectly, rubella or rubeola, is an acute, contagious exanthem, presenting an eruption resembling that of morbilli and throat-symptoms like those of scarlatina. An attack protects from subsequent attacks, but not from measles or scarlatina; neither does an attack of scarlatina or one of measles confer immunity from Rötheln. The *period of incubation* of Rötheln is said to be about fourteen days. The *onset* is usually abrupt. The *temperature* is moderate; the *pulse* not very rapid. In the course of a day or two, there appears, first upon the face and then progressively invading the trunk and the extremities, an *eruption* of small papules usually separated from one another by skin of normal appearance. Sometimes, the intervening skin is erythematous. The eruption lasts for from four days to a week, is attended with itching and is sometimes followed by slight desquamation. The *throat* is usually sore, the fauces reddened and the cervical glands enlarged. *Catarrhal symptoms* are common. The *course* of the disease is usually mild and uncomplicated.

How are Rötheln and scarlatina to be differentiated?

Rötheln is inherently a mild disease; scarlatina is never without gravity. Rötheln lacks the rapid pulse, the high temperature, the "strawberry tongue" and the grave complications of scarlatina. The eruption of Rötheln more nearly resembles that of morbilli than that of scarlatina. Neither Rötheln nor scarlatina is protective against the other.

How are Rötheln and morbilli to be differentiated?

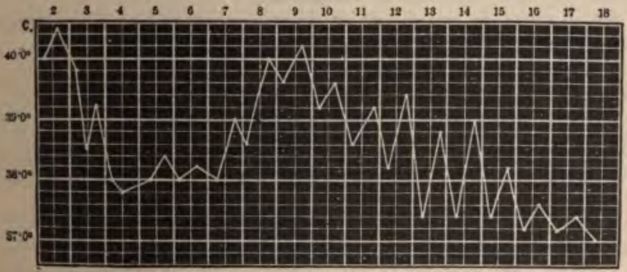
Rötheln resembles morbilli in its rash, in its mildness and in the catarrhal symptoms it presents. The eruption of Rötheln, however, does not show a tendency to crescentic arrangement, while it appears earlier and the individual papules are smaller than is the case in morbilli. Rötheln is milder than morbilli, even as relates to the catarrhal symptoms. An attack of either confers no immunity from the other.

Variola—Smallpox.

Upon what does the diagnosis of smallpox depend?

Smallpox or *variola* is an acute, contagious, epidemic disease, *setting in* with a chill, pains in the back, head and extremities, nausea and vomiting, elevation of *temperature* to 102° or 103° F., with marked exacerbations, increased rapidity of pulse, and a diffuse, *red rash* that is followed on the third or fourth day by the appearance of *papules;* when the temperature declines.

FIG. 13.



Temperature-chart of smallpox. (Strümpell.)

The papules commonly appear first on the lips and forehead. The preceding red rash usually appears first on the arms, and in the neighborhood of the groin. The fauces are apt to be reddened. There may be decided catarrhal symptoms. In the course of the succeeding four or five days, the papules become *vesicles* and the vesicles, in turn, *pustules.* The surface of the pustule is depressed at the middle—*umbilicated.* Each pustule is surrounded by an area of redness, constituting an *areola.* On the eighth or ninth day the pustules rupture and discharge their contents, and the *temperature again rises* (secondary or pyemic fever). (Fig. 13.) Secondary fever is sometimes announced by rigor, and its temperature-course is remittent. The evening temperature reaches from 103° to 105° F. The period of maturation and discharge lasts from three to five days, when crusts form and the temperature declines; in the course of a week the scabs fall off, leaving red cicatrices, which in the course of time become whiter and contracted, leaving "pits."

Ulceration of the larynx and trachea, bronchitis, pneumonia or pleurisy may *complicate* variola. Secondary inflammations are most likely to occur coincidently with the secondary fever. The eye frequently suffers permanent injury as the result of an attack of smallpox. The *period of incubation* of variola is about twelve days.

What are the varieties of smallpox?

Smallpox may be *simple* or *discrete*, *confluent* or *hemorrhagic;* it may assume a *malignant* character.

What are the characteristics of discrete smallpox?

Discrete smallpox is the mildest type of smallpox; the eruption is least profuse, the pustules occurring isolated. Sometimes the pustules are in contact at their periphery, when the disease is said to be *coherent.*

What are the characteristics of confluent smallpox?

In *confluent smallpox*, the pustules are numerous and run into one another. The temperature fails to decline with the appearance of the eruption and is apt to be decidedly high during the period of maturation. The gravity of the case is greater than in discrete smallpox. Typhoid symptoms, delirium, stupor and fatal coma may develop; or death may be brought about by diarrhea, ulceration of the larynx or trachea, endocarditis or other complication.

What are the characteristics of hemorrhagic variola?

In *hemorrhagic variola* the exanthem is from the first constituted of ecchymoses, while hemorrhages may take place from any of the mucous membranes. This is the gravest form. Few cases recover.

What is malignant smallpox?

The epithet *malignant* is sometimes applied to a variety of smallpox, most frequently encountered at the beginning of an epidemic, in which death may occur early in stupor, following delirium, the eruption being ill-defined and perhaps only developed *post-mortem.*

What is varioloid?

Varioloid is smallpox modified by vaccination or by a previous attack of smallpox. The symptoms of the disease are similar to those of smallpox, but milder in degree and shorter in duration; secondary fever is absent. The eruption of varioloid commonly appears on the second or third day; that of variola on the fourth day. The course of varioloid is completed in about fourteen days; that of variola in about twenty-one days.

What is vaccination?

Vaccination consists in the introduction of the virus of cowpock[1] into the lymphatic system of man, usually through the skin denuded of its epithelium, as a protection against variola. The protective influence continues for a period of about seven years, at the end of which time vaccination should be repeated.

---

[1] **What is meant by humanized virus?**

Humanized virus is vaccine-lymph that is not taken directly from the cow (or calf), but from the vaccine-pustule of a human being, usually a child, who may have been inoculated with matter obtained from another child, or from the cow. Unless one is sure as to the purity of the antecedents of the source of the humanized virus, bovine virus is to be preferred.

What is vaccinia?

Vaccinia is the result of the inoculation of cowpock in human beings, and is protective against smallpox. For two or three days following vaccination, little is to be observed, locally or constitutionally. At the end of this time, the site of inoculation presents an appearance of slight redness, which in the next few days becomes intensified, as a vesicle forms and becomes transformed into a pustule, umbilicated and surrounded by a distinct areola. This process goes on for four or five days, when the pustule ruptures and the intensity of the inflammation gradually subsides. In the course of a week or ten days, the crust falls off and leaves a reddish cicatrix, which subsequently becomes white and depressed.

How does smallpox differ from measles?

The eruption of measles is coarsely papular throughout, with a tendency to crescentic arrangement, and is followed by branny desquamation; that of smallpox is irregular in arrangement and passes from the papular into a vesicular and then into a pustular stage, sometimes leaving disfiguring cicatrices. In measles, *after a previous decline in temperature, the appearance of the eruption is attended with renewed elevation;* in smallpox, *the temperature declines with the appearance of the rash* and reascends with the occurrence of pustulation. Smallpox is a grave disease, lasting three weeks; measles is a mild disease, lasting less than two weeks.

How are variola and scarlatina to be differentiated?

Smallpox may be attended with a primary, diffuse red rash, not unlike that of scarlatina, but, at the end of three or four days, papules appear, in turn to be succeeded by vesicles and pustules, finally leaving disfiguring cicatrices. With the appearance of the secondary rash, the temperature declines. The eruption of scarlatina undergoes no change and terminates in desquamation. The temperature is high from the outset and is continuous. Variola is not characterized by the same rapidity of pulse or by the typical "strawberry tongue" of scarlatina. Uncomplicated scarlatina is a disease of less than two weeks' duration; variola rarely lasts less than three.

Varicella—Chickenpox.

What are the characteristics of varicella?

Varicella or *chickenpox* is a mild, contagious disease of childhood, attended with moderate elevation of temperature and the appearance on the first or second day of an eruption of papules, which in turn become transformed into vesicles. The *eruption* appears on the trunk and extremities, on the scalp and face. It comes out in crops and continues for three or four days, the vesicles desiccating and falling off, occasionally leaving cicatrices.

How does varicella differ from smallpox?

Varicella is a mild disease; variola a grave disease. The eruption of smallpox appears on the third or fourth day and passes through papular and vesicular stages to become pustular; that of varicella appears on the first or second day and does not pass beyond a vesicular stage. The appearance of the eruption in smallpox is attended with a fall in the temperature; in varicella, the temperature, rarely high, is uninfluenced by the appearance of the rash. Varicella is a disease of scarcely a week; smallpox is a disease of three weeks. Neither is protective against the other.

How are varicella and varioloid to be differentiated?

There may be a close similarity between the manifestations of varicella and those of varioloid. Varicella is rare in adults because of the immunity conferred by an attack in childhood. Varioloid does not respect age; it is likely to appear in the course of an epidemic of variola in those that have been protected by vaccination. Varicella is the milder affection, and is of the shorter duration. Varioloid is but an attenuation and abbreviation of variola. Both varicella and varioloid are contagious. The existence of parallel cases may be decisive in diagnosis. An attack of the one does not protect against invasion by the other.

How are varicella and morbilli to be differentiated?

The eruption of measles is coarsely papular; it appears on the third or fourth day and displays a special proclivity to invade the face, with a tendency to crescentic arrangement. The eruption of chickenpox is at first papular and subsequently vesicular; it appears on the first or second day and is rather less than more abundant on the face than on the trunk. The catarrhal symptoms of measles are wanting in varicella. The temperature-course is not characteristic in varicella, as it is in measles.

Erysipelas.

What are the symptoms of erysipelas?

Erysipelas is an acute, infectious disease, *setting in suddenly* with a *chill*, sometimes with nausea and vomiting, or with convulsions, followed by considerable *elevation of temperature*, and the appearance, usually at some part of the face, and in most instances near one or other ear, of an area of *red, brawny induration.* The redness progressively increases in extent, is *definitely circumscribed* by an elevated line of demarkation, and is attended with swelling.

The *appetite* is impaired. The *tongue* is coated.

The *urine* is usually albuminous, and often contains tube-casts.

There is often some soreness of the throat, and in some cases the disease may extend through the nasal passages into the pharynx and larynx, causing grave complications, and, perhaps, fatal edema. Sometimes the disease begins in the throat.

Involvement of a cerebral sinus may occur, giving rise to severe symptoms and eventually causing death.

Usually, in the course of a week or ten days, the redness and swelling subside, desquamation sets in, and the temperature gradually reaches the normal.

In some cases, the disease displays a *migratory* tendency, (*erysipelas migrans*), continuing for a long period of time, and in turn appearing at various parts of the body. In children, erysipelas sometimes appears first in the neighborhood of the *anus.* The disease often attacks *wounds.*

What are the differential features between erysipelas and scarlatina?

Scarlatina is especially a disease of childhood. Erysipelas is more common in adults than in children.

The temperature is high in both, but the rapidity of the pulse is the more characteristic of scarlatina.

The rash of erysipelas is circumscribed in extent, limited in area, homogeneous in color; it usually appears upon the face, and presents a peculiar brawny induration, with well-defined borders; while the eruption of scarlatina is extensive in distribution and punctate in character.

An attack of scarlatina confers immunity from subsequent attacks; one attack of erysipelas rather predisposes to the occurrence of subsequent attacks.

Of the two, scarlet fever is the longer in duration.

How does erysipelas differ from simple erythema?

Simple erythema presents a diffuse redness, of transitory character, without febrile concomitants or sequelæ.

Erysipelas is a serious affection, with considerable elevation of temperature and characteristic rapidity of pulse. It lasts a week or more, is followed by desquamation, and is intimately related with nephritis.

How are facial erysipelas and herpes zoster of the forehead and face to be differentiated?

Facial erysipelas and herpes zoster of the forehead and face present a number of symptoms in common. That which distinguishes the latter, however, is that the eruption begins as a number of vesicles and does not extend beyond the middle line. The pain is much greater in herpes than in erysipelas. The constitutional symptoms are more profound in erysipelas than in herpes.

How are variola and erysipelas to be differentiated?

When variola is attended with a primary roseola, the disease may, for several days, simulate erysipelas. The redness of erysipelas, however, is distinctly circumscribed, although it may slowly spread, and is attended with brawny induration, while that of smallpox rapidly spreads from the face to the trunk and extremities. On the third or fourth day, if the disease is variola, papules appear, progressively passing through the stages of vesicles and pustules. The eruption of erysipelas undergoes little change, unless large blebs form, until on about the fifth or seventh day, when it may subside with desquamation.

Dengue.

What are the symptoms of dengue or "break-bone fever?"

Dengue is an exanthematous and arthritic disease of hot climates, occurring in *epidemics*, and having a *period of incubation* of about four days. It is characterized by severe pains in the muscles and joints, which may be stiff and swollen. The knees are especially prone to be affected, so that the gait presents a peculiar character. The disease may be gradual in *onset*, with anorexia, headache, vertigo, drowsiness, or it may set in suddenly with a chill, moderate elevation of temperature and the appearance of an erythematous rash. There are stiffness of the neck and pain along the spine and in the lumbar region. The *fever* reaches its acme within twenty-four hours. The *temperature* fluctuates, rising and falling, but in the course of two or three days subsides nearly or quite to the normal; the eruption disappears, though constitutional depression and more or less pain continue. As a rule, nausea and vomiting do not occur at this stage, though the tongue may be heavily coated, and there may be other symptoms of gastric irritability.

After an intermittence of from forty-eight to seventy-two hours or more, the fever returns and a new eruption appears, as a rule resembling the eruption of scarlatina. Sometimes the eruption is more like that of measles, or it may be urticarious or vesicular. It is attended with heat and itching. Nausea and vomiting are usually manifested with this renewal of the fever.

In the course of a few days, desquamation occurs; convalescence sets in, but is tardy and protracted; there is weakness and more or less rheumatoid muscular pain; lymphatic swellings in the neck, groin or axilla often appear during the febrile period or during convalescence. When the disease invades a community, few escape.

How does dengue differ from scarlatina with arthritic manifestations?

In dengue the fever is not continuous and pursues a course different from that of scarlatina; the arthritic symptoms are of earlier occurrence, and the pain is of a characteristic nature, giving the name "break-bone" fever. The eruption develops much later, and is sometimes quite different in appearance from that of scarlatina. The erythematous rash of the period of invasion is slight, inconstant and disappears without desquamation when the remission or intermission occurs. Throat-symptoms are not common.

In what respect does dengue differ from influenza?

Influenza is the graver disease; its symptoms are the more intense.

Catarrhal symptoms are frequent in influenza, infrequent in dengue.

Eruptions are exceptional in influenza, the rule in dengue.

Joint-pains are more decided in dengue. Hyperesthesia of the cutaneous surface is more common in influenza.

The course of dengue is interrupted by a remission. The course of influenza is usually though not invariably continuous to its termination.

Influenza is independent of climate. Dengue prevails only in certain localities. Convalescence is even more tedious and protracted from dengue than from influenza.

Diphtheria.

What are the symptoms of diphtheria?

Diphtheria is an acute, infectious disease affecting children especially, but adults as well, and characterized by septic fever with superficial coagulation-necrosis of mucous membranes, especially of those of the pharynx, larynx and nares.

The attack may *begin* insidiously, or *set in* suddenly with a chill, followed by considerable fever.

Pain in the throat in swallowing may or may not be complained of. The fauces will be seen to be livid, the tonsils usually swollen.

Soon, there appear, in greater or less extent and rapidly spreading over the tonsils, the half-arches, the uvula and the posterior wall of the pharynx, grayish or yellowish patches of false membrane, the forcible detachment of which is followed by bleeding. There are enlargement of the submaxillary and cervical glands and tumefaction of the soft tissues of the neck, externally.

The *constitutional disturbance* becomes profound. Albuminuria is common. From the pharynx the inflammation and necrosis may extend to the larynx and to the nose, and to the difficulty of swallowing are added croupy cough, aphonia, difficulty of breathing and a nasal discharge. The diphtheritic process is sometimes primary in the nose, and may thus escape detection, unless careful examination be made. From the larynx the false membrane may invade the trachea and bronchi. Pneumonia may occur. The action of the *heart* becomes weak and often intermittent. Septicemia, heart-failure and suffocation from obstruction of the larynx or bronchi are the common causes of death.

The *fatality* of the disease varies in different epidemics. Even in times of grave epidemics, there are many mild cases that become foci of infection. *Chronic diphtheria* of the throat is not so rare as it is commonly considered to be and is likewise a focus of infection. Paralysis from peripheral neuritis or cerebral thrombosis may be a *sequel.*

The *palsy* of diphtheria may appear in the course of the disease, but it is more common after the acute attack is at an end. Adults are rather more prone to suffer than children. The most common manifestations are *paralysis of the palate*, permitting regurgitation of fluids and giving rise to nasal speech; *paralysis of the ciliary muscle* (cycloplegia), causing loss of power of accommodation; loss of *knee-jerks.* There may be more general palsy, with deranged sensation, ataxia and trophic changes.

How are diphtheria and scarlatina to be differentiated?

At the onset, the diseases may be indistinguishable. Both present the evidences of constitutional disturbance, with localized throat-symptoms. Possibly, the pulse may be relatively more rapid in scarlatina than in diphtheria. In from twenty-four to thirty-six hours, however, the appearance of a scarlet rash, as well as the subsequent course of the disease, dispels all doubt.

In diphtheria, the symptoms centralize themselves about the throat; in scarlatina the throat disturbance represents but a part of the general derangement. The paralyses commonly seen after diphtheria are rare after scarlatina. More common during the course of scarlet fever or subsequently are suppurative ear-disease, nephritis and glandular enlargement.

Diphtheria and scarlatina may coëxist in the same patient.

How is tonsillitis to be distinguished from diphtheria?

Deposits on the tonsil may appear diphtheritic. They show little or no tendency to spread, however. Extension is characteristic of the diphtheritic membrane. In lacunal tonsillitis, the discreteness of the plugs and their situation at the orifices of the ducts are characteristic, and their creamy color is different from that of the diphtheritic pseudomembrane. Microscopically, they will be seen to be made up of desquamated epithelium, of sebaceous material and of ordinary fungi. The diphtheritic membrane is constituted of meshes of fibrin containing necrotic tissue. In herpetic tonsillitis the eruption first appears as papules that soon become vesicles; but it is rarely seen at this stage; when ulcers and fibrinous deposits form and become confluent, the discrimination is difficult. Still, the herpetic patch is quite superficial, and more readily detached, leaving less erosion and causing less hemorrhage in its separation than does the diphtheritic patch. The former is usually the less extensive, and here and there, perhaps, the circular form of an isolated ulcer may give evidence of its origin. If necessary, inoculation-experiments and bacteriologic investigation may also help to discriminate. The constitutional symptoms of tonsillitis are less profound than those of diphtheria; local subjective symptoms, such as soreness, odynphagia and burning, are usually the more intense in tonsillitis, which is not, as a rule, followed by paralysis of the palate. Albuminuria is not usual.

How is membranous croup to be distinguished from diphtheria?

Until the physician acquires sufficient experience to warrant a personal opinion, he had best consider all cases of membranous croup diphtheritic. The discrimination is difficult and disputed.

How are diphtheria and stomatitis to be differentiated?

The deposits in stomatitis are seated upon the mucous surface of the lips and cheeks and upon the tongue, while the membrane of diphtheria is usually seated in the pharynx, from which, as a center, it is distributed. The constitutional derangement is not as profound in stomatitis as in diphtheria. Stomatitis readily yields to mild general and local measures, while diphtheria is more rebellious to treatment. The fatality and the severe sequelæ of diphtheria are wanting in stomatitis.

Glanders—Farcy—Equinia.

What are the clinical features of glanders?

Glanders, *farcy* or *equinia* is an infectious disease, especially peculiar to horses, asses, and mules, from which it is transmitted to man through abrasions of the skin and through the mucous surfaces of those that come in contact with the diseased animals.

The site of inoculation displays evidence of active inflammation; especially is this marked in the nasal passages. There are also malaise, headache, elevation of temperature and pains in the limbs; the urine may be albuminous. Soon there appears a macular eruption, which becomes vesicular, then pustular and finally umbilicated. The pustules may rupture and leave ugly ulcers. In addition, nodules form beneath the skin; these also soften and may rupture, discharging sanious pus and detritus. Another characteristic symptom of glanders is ozena. There is at first a moderate, thin discharge from the nostrils, soon, however, becoming profuse and purulent. The mucous membrane of the nares and contiguous structures is involved in intense inflammation and may become ulcerated. Catarrhal pneumonia and purulent arthritis are occasional complications. Glanders may be transmitted from man to the lower animals by inoculation.

How are glanders and variola to be differentiated?

In glanders, there may be a history with the local evidences of inoculation with the specific virus of the disease. The eruption of variola does not appear until the third or fourth day; with its appearance the temperature falls. The eruption of glanders may appear within the first twenty-four or forty-eight hours of the disease; it reaches a pustular stage much earlier than that of variola; there is no decline of temperature with its appearance. The eczema and the subcutaneous nodules of glanders are wanting in variola.

How are the ozena of glanders and that of syphilitic disease to be differentiated?

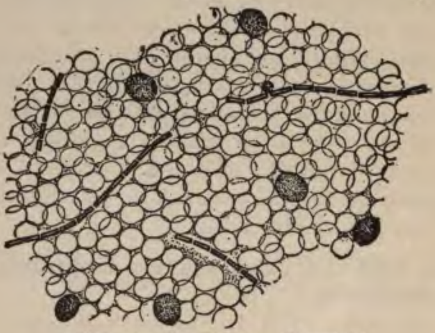
Ozena is dependent upon destruction of the nasal structures and putrefactive decomposition of the secretions. It is thus not distinctive of a single disease. Occurring in syphilis, it is a late manifestation, and probably has been preceded by well-defined symptoms. As seen in glanders, it occurs at the height of the disease, and is associated with a pustular eruption and the presence of nodules beneath the skin. The mode of infection differs in the two diseases.

Anthrax—Wool-sorters' Disease.

What are the clinical features of anthrax?

Anthrax, *wool-sorters' disease*, *charbon*, *malignant pustule* or *splenic fever* is an infectious disease, due to inoculation with the bacillus anthracis (Fig. 14.) It develops in butchers, wool-sorters, workers in hides, stevedores and others that, with cut or wounded or abraded hands, manipulate the wool or skins of animals that have died of splenic fever or charbon. The infection sometimes gains entrance through a scratch on the cheek or an abrasion of the lips. In butchers, the tongue is sometimes infected from a knife taken between the teeth. In those that carry hides upon their shoulders, the neck may be the site of local infection; these cases are likely to be more than ordinarily dangerous. The disease may apparently result from eating the flesh or from drinking the milk of infected animals.

FIG. 14.



Anthrax bacilli in blood. (Vierordt.)

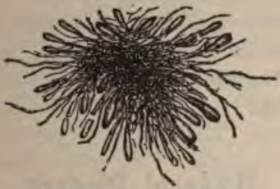
At the site of inoculation, a pimple appears; the skin in its neighborhood becomes red and infiltrated; the papule becomes vesicular and pustular, with subsequent gangrene; other vesicles or pustules form and also become gangrenous; there results a characteristic eschar, which presents the appearance of an elevated patch, consisting of a zone of low, whitish vesicles surrounding a depressed brownish, purplish, or black center, with an outer zone of red induration. Beyond this is usually a region of swelling and edema of variable extent. The spleen and the lymphatic glands in communication with the infected regions enlarge. There is often considerable local tenderness. The *constitutional symptoms* are those of septicopyemia: malaise, headache, depression, fever. According to the mode of introduction of the poison, or the direction in which infection spreads, other manifestations appear. Sometimes the gastro-intestinal tract appears to bear the brunt of the disease and there are nausea, vomiting, abdominal pains and diarrhea, the stools being bloody. Death may take place from exhaustion or from septicemia. At other times, thoracic symptoms predominate. There are then dyspnea, a sense of oppression of breathing, hemoptysis and cyanosis. Death may take place from edema of the larynx or of the mediastinum. Characteristic bacilli may often be found in the blood, pus, sputum, feces, or urine. Under proper treatment recovery frequently takes place.

Actinomycosis.

What is actinomycosis?

Actinomycosis is a condition dependent upon the presence of ray-fungi: *actinomyces bovis.* (Fig. 15.) The disease is more frequent in drovers and in those that have to do with cattle, from which the parasite, as found in man, is usually derived. The cattle become infected through their food. The fungus gains entrance through a breach in continuity of the surface and, finding its way to a suitable nidus, gives rise to the formation of a seropurulent collection; this manifests itself as a tumor that usually finds vent externally. In the matter discharged, yellowish miliary nodules, composed of fungi, can be detected. The lower jaw seems to be a favorite seat of the disease, infection taking place through decayed teeth; sometimes extensive destruction of bone results. At other times, purulent collections form in internal viscera. When the pleura is infected the ribs may suffer severely.

FIG. 15.



Actinomyces. (Ziegler.)

Foot-and-Mouth Disease.

What is foot-and-mouth disease?

Foot-and-mouth disease is a rare affection that occurs in sheep, cows, pigs and horses, and that occasionally seems to be transmitted to man. It manifests itself by the appearance of vesicles and bullæ in the mouth and on the feet at the margins of the hoofs and, in cows, on the udder and teats. The disease may be transferred directly to man by inoculation—thus to the butcher or to the veterinary surgeon, or it may be conveyed by milk. In man, vesicles form in the mouth, on the face, on the hands and on the feet. In the course of two or three days, the vesicles rupture, discharging opaque, yellowish fluid, and leaving dark-red ulcers. There are also fever, loss of appetite, pain in eating, swelling of the tongue, fetor of the breath, salivation and derangement of digestion. In children the disease may prove fatal.

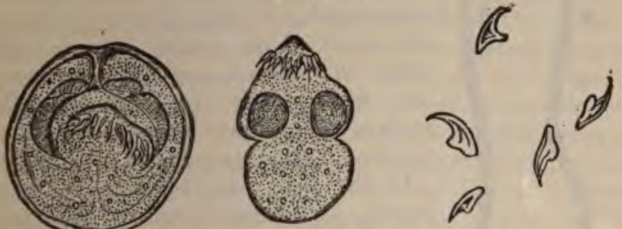
Hydatid Disease.

What is hydatid disease?

An *hydatid cyst* is a parasitic formation due to ingestion of the ova of the *tenia echinococcus*, the tape-worm of dogs, in which it is derived from the flesh of sheep, or pigs, or less frequently, kine, suffering from hydatid disease.

When the ovum enters the stomach of man, its capsule is dissolved, and the immature embryo or scolex (Fig. 16) is set free to continue its migrations. Arriving at its destination, the irritation to which it gives rise results in the formation of a membranous envelop, in which the parasite continues its development. This capsule, and its contents together constitute an *hydatid cyst*. An hydatid cyst contains within the capsule, a vesicle or mother-sac, consisting of concentric layers of a gelatinous material, inclosing the embryo and more or less fluid. The fluid is clear, opalescent, and faintly alkaline. Within this develop other similar sacs, so-called daughter-vesicles, and within these again, granddaughter-vesicles; the mother-sac and its investing membrane continue to enlarge, if in a favorable situation, until ultimately the cyst attains an enormous size. Multiple cysts may form. The daughter-vesicles contain a germinating layer that produces new scolices. These consist of a head, four suckers and a row of hooklets. The discovery of the hooklets (Fig. 16) in fluid removed from the cyst is diagnostic. *Sterile echinococci* or *acephalocysts* do not produce scolices.

Fig. 16.



Tenia Echinococcus—vesicle, scolex and hooks. (After Heller.)

Hydatids may develop in various viscera, but are most common in the liver.

Trichiniasis.

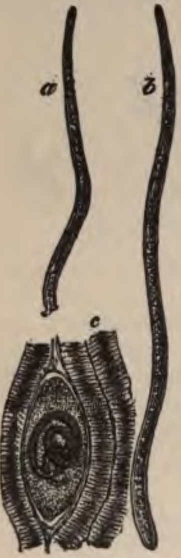
What is trichiniasis?

Trichiniasis is a disease set up by the *trichina spiralis* (Fig. 17), a small roundworm that finds its way into the intestine with meat obtained from diseased swine.

In the intestine the mature female throws off embryos, which pass through the walls of the intestine and into the blood-current, finding their way into the voluntary muscles in different parts of the body. Here the embryos occasion irritation and inflammation, so that about each a capsule forms in which lime-salts are in time deposited; in this way the death of the embryo may be brought about.

What are the symptoms of trichiniasis?

FIG. 17.



Trichina. *a*, Male; *b*, Female; *c*, Muscle-Trichina. (V. Jaksch)

The symptoms of trichiniasis present themselves in three *stages*. In the *first*, which lasts about a week, the trichinæ are undergoing development in the alimentary canal, as a result of which the symptoms of gastro-intestinal derangement appear. In the *second stage*, lasting two or three weeks, the embryos pass from the intestine into the muscular tissue. Finally, retrogressive changes take place in and around the trichinae encapsulated in the muscles.

In the *first stage*, the appetite is impaired; the tongue is coated, the breath is foul; there are malaise, nausea, a bad taste, diarrhea, abdominal pain and slight fever.

In the *second stage*, there is edema, apparent in the face and sometimes extending downwards; there are also pains in the muscles, which are swollen; defects of the ocular muscles and of accommodation sometimes appear; the senses and various functions may be affected; sleep is disturbed and there is moderate fever. The muscles are sensitive to touch, which may detect the minute nodules. Death may result from exhaustion, pneumonia or ulceration of the bowel. In favorable cases, the symptoms gradually subside and the patient enters upon the *third stage* of the disease. Active symptoms are now in abeyance, but there may be some stiffness of the muscles, while nodules in the muscles may be detectable.

Ultimately, however, the patient may be restored to a fair degree of health.

Acute Rheumatism—Rheumatic Fever.

What are the symptoms of acute rheumatism?

Acute rheumatism or *rheumatic fever* usually follows exposure to cold. An almost identical condition is sometimes observed in the course of puerperal fever and as a sequel of scarlatina. It is thought to be dependent upon the presence of lactic acid in the blood. One of the larger *joints* becomes painful, enlarged, hot and reddened. It is evidently inflamed; sometimes the presence of fluid can be detected. Soon, another joint, probably the corresponding joint on the opposite side, or the next contiguous joint, becomes similarly involved; and in this way the process may extend, until most or all of the large joints are in turn attacked. On account of the pain, the patient is immovably helpless. From the surface of the body exudes an acid sweat. The *temperature* ordinarily ranges from 102° F. to 104° F., rising with each fresh access of joint-symptoms, and declining gradually with the termination of the disease. The pulse is disproportionately frequent; it may be full and bounding. The *urine* is scanty, high-colored, and quite acid; it may contain a trace of albumin.

In many cases, to the articular manifestations *endocarditis* is added, as a result of which permanent valvular lesions may be established. Among other *complications* are inflammations of the pericardium, the pleura, the peritoneum, the kidney and, though rarely, the cerebro-spinal meninges. As a rule, delirium is due to the toxemia and not to meningitis.

Sometimes the temperature rises even to *hyperpyrexia*, and there are decided cerebral symptoms—delirium, convulsions, coma, death.

Untreated, the *duration* of acute rheumatism is about six weeks; sometimes much longer. Relapses are not uncommon. An attack predisposes to subsequent attacks.

Purpura is sometimes seen in the course of rheumatism; sometimes tuberculated cutaneous nodules.

In some cases of rheumatism, hereditary influences can be traced. Some authorities consider the disease a dyscrasia, others a neurosis. There appears to be an indefinable, yet close relation between acute rheumatism and chorea.

Articular rheumatism not uncommonly accompanies, precedes or follows acute tonsillitis. In such cases, pericarditis and endocarditis may sometimes be detected if carefully searched for.

How are pyemia and acute rheumatism to be differentiated?

Both pyemia and acute rheumatism occasion arthritis, sweats, cardiac complications and cerebral symptoms. Periodicity and rigors, as well as metastatic invasion of internal structures remote from the primary seat of disease, which are common in pyemia, are wanting in rheumatism. The constitutional depression is more profound in pyemia than in rheumatism. In one, an obvious or obscure focus of suppuration exists; in the other, there is a history of rather abrupt onset following exposure to cold or wet.

How are acute synovitis and acute rheumatism to be differentiated?

Acute synovitis usually involves but a single joint; it is characteristic of acute rheumatism to progressively attack many joints. The constitutional phenomena are more profound in acute rheumatism than in acute synovitis.

The peculiar, acid sweats, as well as the cardiac complications of rheumatism are not seen in synovitis.

In duration, synovitis is the shorter disease.

Gonorrheal Synovitis.

What is gonorrheal synovitis?

Occasionally, in the course of an attack of gonorrhea, a large joint—and usually but one joint, such as the knee, the elbow, the wrist, the ankle or the shoulder, becomes tumid, painful, tender and hot, and the adjacent textures may be edematous. The urethral discharge often ceases with the appearance of the synovitis. In successive attacks of gonorrhea, different joints may be involved. Permanent stiffness and impaired mobility constitute a common sequel. Cardiac complications have been observed in some cases of gonorrheal synovitis. The diagnosis depends upon a knowledge of the existence of a specific urethritis. Sometimes, the course of the temperature is suggestive of a pyemic or of a septic condition.

Syphilitic Arthritis.

What is syphilitic arthritis?

Every now and then, in the course of syphilis, one or more joints become involved in inflammation, with all of the characteristics of an arthritis. The discrimination of the condition depends upon a recognition of its association with syphilis. As a rule, cardiac complications are wanting in syphilis, and the arthritis is more strictly limited to one, or at most, two joints, the migratory tendency of acute articular rheumatism being absent.

Subacute Rheumatism.

What is subacute rheumatism?

At times, as a result of exposure to cold and wet, muscular movement becomes painful, in consequence of a rheumatic involvement of the muscle-sheaths. The patient sometimes mistakes for paralytic weakness the restraint of motion by pain. The pain is also in some degree spontaneous and influenced by meteorologic conditions. Affecting the muscles of the lumbar region, the condition is termed *lumbago*. Affecting the muscles of the neck it may give rise to *torticollis* or *wry-neck*.

How are subacute rheumatism and neuralgia to be differentiated?

Both subacute rheumatism and neuralgia occur in paroxysms, superinduced by suitable meteorologic conditions. Rheumatism is more common in men; neuralgia in women. The pain of the former is rather dull and diffused; that of the latter sharp and confined to the distribution of an affected nerve, in the course of which may be found several tender points. Rheumatic pain more commonly than neuralgic pain is aggravated by movement.

How are subacute rheumatism and trichiniasis to be differentiated?

In trichiniasis, in addition to symptoms simulating those of subacute rheumatism, there are evidences of a cachexia, wasting, debility and symptoms of gastro-intestinal derangement, with a history of the ingestion of diseased meat. At an advanced stage of the disease, it may be possible to detect the nodules to which the encapsulated parasites give rise.

Myalgia.

What is myalgia?

As a result of muscular strain, groups of muscles become painful to touch and on movement, in association with some degree of cutaneous hyperesthesia.

Chronic Rheumatism.

What are the symptoms of chronic rheumatism?

As a result of an attack or of repeated attacks of acute rheumatism, numerous joints in different parts of the body remain enlarged, stiff and painful. Sometimes the condition is insidious, progressive and chronic from the outset. However produced, the functions of the various joints are impaired; attacks of pain occur and are apparently influenced by meteorologic conditions. Wasting of the muscular structures adjacent to the diseased joints takes place.

Chronic rheumatism may affect both joints and muscles, or the muscle-sheaths or tendons only (*muscular rheumatism*), or it may attack the nerve-sheaths (*rheumatic neuralgia*). The principal symptoms of muscular and of nerve-rheumatism are pain, spontaneous and on motion, with accompanying tenderness, usually localized.

How are the enlarged joints of chronic rheumatism to be distinguished from those of chronic spinal disease?

Trophic changes in the large joints—enlargement, effusion, subluxation, arthropathies—take place in the course of some chronic spinal affections, notably posterior spinal sclerosis. As a rule, but one or a few joints are involved. In chronic rheumatism, many joints are involved. In case of disease of the spinal cord ordinary scrutiny should detect the existence of symptoms indicative of such a condition.

Acute Gout.

What are the symptoms of acute gout?

Acute gout is a recurrent paroxysmal affection thought to be dependent upon the presence of an excess of uric acid in the blood. It occurs chiefly in those of a sedentary or inactive mode of life, who indulge excessively in the luxuries of the table, more especially in meats, sweets, sweet wines and malt liquors. Those that have been active in out-door sports and afterwards, while diminishing their exercise, maintain the heavy diet formerly appropriate, are extremely liable to gout. The tendency to gout is distinctly hereditary, and in some cases of marked gouty diathesis the attacks may occur despite personal abstemiousness.

The *paroxysm* may be brought on by an unusual excess, by a fit of anger, by worry or anxiety or by exhaustion. Its *advent* is sometimes unannounced; at other times, it is preceded by symptoms of indigestion, by mental irritability or depression. The attack usually *sets in* suddenly at night, the patient being awakened by intense pain most commonly referred to the metatarso-phalangeal joint of the great toe. There is *fever* in proportion to the intensity of the local affection. The pain moderates somewhat towards morning, when the patient falls into a gentle perspiration and is again able to sleep. Towards night the pain returns. The joint is now noticed to be tender, red, swollen and edematous; finally desquamation takes place. Other joints are successively involved, the morbid process showing an affinity for the smaller articulations. The attack gradually subsides, leaving the affected joints a little stiffened and swollen. At the height of the attack the proportion of uric acid in the blood is increased, while that excreted in the urine is diminished. When the paroxysm is over the quantity of uric acid in the urine is increased. In an attack of acute gout, the joint-symptoms may suddenly subside, and gastric, cardiac or even cerebro-spinal symptoms be substituted.

Sometimes the attack is manifested from the first by visceral rather than by articular crises. Visceral crises are more likely to occur late in the history of the case than early in its course, and they sometimes prove fatal.

Chronic Gout.

How are acute rheumatism and acute gout to be differentiated?

Gout is an hereditary affection, occurring in paroxysms, in which the first metatarso-phalangeal articulation and other small joints are involved. In acute rheumatism, a history of heredity is frequently wanting; the large joints are especially involved. The duration of an attack of rheumatism is many weeks; an attack of gout subsides in the course of a week or two. The uratic deposits of gout are wanting in rheumatism. The sweats of rheumatism are absent from gout. Cardiac complications are common in acute rheumatism; gout never occasions endocarditis, but chronic interstitial nephritis is a common sequel.

What is chronic gout?

In those that have had a number of paroxysms of acute gout, or sometimes chronically from the first, deposits of urates take place around the diseased joints, in the articular cartilages and elsewhere, as in the lobe of the ear, in the kidneys and in the spleen. As a result there is painful thickening of the affected articulations, which are stiff and finally become deformed. Sometimes distinct "chalk-stones" may be felt, and in extreme cases these may cause ulceration and appear externally. Gout is a potent cause of arterio-capillary fibrosis. An excess of fibrous tissue develops in the viscera and in the walls of the blood-vessels, with secondary contraction. Chronic interstitial nephritis is a common sequel.

Lead-poisoning may give rise to lesions exactly resembling those of chronic or of subacute gout.

Lithemia.

What are the clinical features of lithemia?

Lithemia is modified gout—a manifestation of the uric-acid diathesis. It is caused by defective oxidation within the body and is dependent upon imperfect tissue-metabolism.

Lithemia manifests itself by varied symptoms, among which are sallowness or abnormal redness of complexion, impaired or perverted appetite, a metallic taste, deranged digestion, constipation, headache, vertigo, irritability of temper, a tendency to melancholia, abnormal drowsiness or sleeplessness, palpitation of the heart, irritative cough, disturbances of vision, noises in the ears, anomalous cutaneous eruptions, transient localized edema, and cramps in the calves of the legs. *Micturition* is frequent and burning, the *urine* usually being diminished in quantity and containing an excess of uric acid and urates; phosphates and calcium oxalate are likewise frequently in excess; in cases attended with paroxysmal flushing red blood-corpuscles and albumin are occasionally found; sometimes a small number of tube-casts. Chronic catarrhal hepatitis and functional inactivity of the liver are frequently—perhaps causally—associated with lithemia. Fibrous degeneration of the kidneys and of the walls of the smaller arteries, with cardiac hypertrophy, may be an ultimate *sequel.*

For what affections may lithemia be mistaken?

Unless in a given case one bears in mind the possibility of the existence of lithemia, and is on the alert for its detection, the condition may be mistaken for almost any functional disorder, or even for serious organic disease of the heart, brain, stomach, intestine, or other organ.

The discrimination depends partly upon the exclusion of visceral lesions, and partly upon the results of urinalysis: the finding of an excess of urates or free uric acid pointing to the existence of lithemia. An hereditary tendency to gout or rheumatism, or the existence of gout, rheumatism, or diabetes in other members of the patient's family, or the fact that the patient's habits of life are such as are likely to lead to the development of gout, should direct attention to the probability of the existence of lithemia.

It must not be forgotten that fibroid changes in the blood-vessels and kidneys are frequent concomitants of the uric-acid diathesis.

Rheumatoid Arthritis—Arthritis Deformans.

What are the clinical features of rheumatoid arthritis?

Rheumatoid arthritis, *arthritis deformans*, often incorrectly called *rheumatic gout*, is a morbid condition in which destructive changes take place in many joints of the body, resulting in thickening, impairment of mobility, deformity and pain. The articular cartilages undergo softening and absorption; the ends of the bones become enlarged and sclerotic, while the apposed surfaces become smooth from mutual pressure; the subjacent bone, however, becomes rarefied and brittle; lime-salts are deposited in the remains of the articular cartilages. A peculiar crepitus due to the apposition of roughened surfaces can often be elicited on manipulation of the affected joint. At a later stage, eburnation takes place, and the apposed bony surfaces slide over one another with abnormal facility.

The disease manifests a tendency to symmetrical involvement. When the hands are involved, a peculiar deformity results—the fingers being deflected towards the ulnar side. Occasionally, hard fibrous nodules are found in the muscles at a short distance from the affected joints.

The *onset* of the disease is usually insidious; occasionally it is acute and attended with febrile symptoms, with pain, with swelling and with redness of the affected joints.

Arthritis deformans occurs in those exposed to unfavorable hygienic influences, in the weak and ill-fed, in those exhausted by frequent childbearing, by prolonged lactation, by grief or by anxiety.

The disease is not directly fatal. It occasions no cardiac complication. When the larger joints (especially the hip, knees and elbows) are involved there often results decided *muscular atrophy*.

How are chronic rheumatism and rheumatoid arthritis to be differentiated?

In rheumatism, the larger joints of the body are especially involved; rheumatoid arthritis involves the smaller joints as well. The deformity of rheumatism is essentially dependent upon a hyperplasia of the fibrous structures that enter into the formation of the articulation; the joint-lesions of rheumatoid arthritis are partly destructive in character and occasion peculiar deformities of the hands and feet, while irregular exostoses form on the articular extremities of the bones. The tendency to symmetrical invasion is more conspicuous in rheumatoid arthritis than in chronic rheumatism. The latter is usually a disease of advanced life; the former may appear in early adult life.

How are gout and rheumatoid arthritis to be differentiated?

Rheumatoid arthritis lacks the paroxysmal character of gout. Unlike gout, it is observed in the underfed rather than in the overfed. In rheumatoid arthritis the deposits of uric acid and of urates in various structures, characteristic of gout, are wanting. Gout never presents the peculiar deformities of the hands and feet seen in rheumatoid arthritis. The latter does not lead to the fibroid condition of the kidneys, heart and vessels to which gout gives rise.

THE BLOOD.

What are the methods of studying the constitution of the blood?

For purposes of diagnosis especially, the blood is frequently examined as to its *corpuscular richness*, as to the *proportion of hemoglobin* it contains, and as to the presence of *abnormal* bodies.

The *corpuscular richness* of the blood is determined by means of an instrument called a *hemocytometer* or *blood-cell counter*, which consists of a shallow cell of known capacity, mounted on a glass slide, in microscopic divisions of which the numbers of red and white blood-corpuscles contained in a centesimal dilution of blood are respectively counted. In healthy men, the blood contains about five million red corpuscles to the cubic millimeter; in women, about four and a half millions. The number of white cells is held not to permanently exceed under normal conditions, 10,000 per cubic millimeter. The normal ratio of white cells to red cells varies between 1 : 400 and 1 : 800.

The *proportion of hemoglobin* in the blood is determined by comparing the color of the diluted blood with a standard, called a *hemoglobinometer*, the result being expressed in percentages.

The percentage of hemoglobin is sometimes spoken of as *absolute* or *total;* sometimes as *relative.* By the term *absolute percentage* is meant the comparative richness in hemoglobin of the whole volume of blood, as shown by the direct reading of the hemoglobinometer scale. By *relative percentage* is meant the relation or ratio of the absolute hemoglobin percentage to the percentage of red corpuscles. Thus, if in a given case, the number of red corpuscles to the cubic millimeter is estimated at 3,000,000, or 60% (5,000,000 being taken as the standard or 100 per cent.), and the hemiglobinometer reading is 54 per cent., the latter figure (54%) would represent the absolute hemoglobin percentage, while the relative hemoglobin percentage would be $\frac{54}{60}$ or 90 per cent.

Deficiency of blood is known as *anemia* or *oligemia*; deficiency of corpuscles, as *oligocythemia;* deficiency of hemoglobin, as *oligochromemia;* deficiency in solid constituents of the plasma, especially albumin, as *hydremia;* temporary excess of white corpuscles, as *leukocytosis;* persistent excess of white corpuscles, as *leukemia* or *leukocythemia.* The normal diameter of the red blood-corpuscle is 7μ. Smaller red corpuscles are called *microcytes;* larger, *megalocytes.* Irregularly-shaped red corpuscles are called *poikilocytes.* Certain white cells that readily absorb the staining material called eosin are from this fact termed *eosinophile* cells.

The blood sometimes contains *parasites*, such as the hematozoa of malaria, the spirilla of relapsing fever, the bacilli of anthrax, the embryos of filaria sanguinis hominis, etc.

Anemia.

What are the symptoms of anemia?

Simple anemia may be a *result* of hemorrhage, of long-continued discharges, of syphilis, malaria, fevers and wasting diseases, of mal-assimilation, of impaired nutrition, of the presence of parasites or of poisons in the system.

The *blood* is *deficient* in *quantity* and in *quality.* The actual volume of the circulating fluid, as well as its corpuscular richness, is diminished. The number of *red cells* and the number of *white cells* are less than normal; so is the absolute quantity of *hemoglobin*, while the relative proportion per corpuscle may be scarcely altered. Some of the red cells are ill-shaped and diminutive.

Anemia long continued gives rise to fatty degeneration of various structures, notably of the walls of the bloodvessels, of the heart and of other viscera.

The *countenance* and visible mucous membranes are usually pale; though in exceptional instances of great vascularity of the face the complexion may be rosy. The eyeball has a bluish tint. There are shortness of breath, an undue readiness of fatigue, and a disinclination to mental or physical effort. The patient complains of neuralgia, of headache, of vertigo and of sleeplessness. The *appetite* and *digestion* are impaired; constipation is the rule. The *urine* is pale and may be of low specific gravity, from diminution in urea. Emaciation is not always evident. The action of the *heart* is enfeebled. Palpitation is common. The *pulse* is soft, compressible, and usually small. A soft, blowing murmur is often to be heard at the base of the heart and in the vessels of the neck; in the arteries less constantly than in the veins (*venous hum*, "*bruit de diable*"). Edema ultimately develops and hemorrhages from various surfaces may take place.

When anemia is associated with enlargement of the spleen it is termed *splenic anemia.* By some authorities splenic anemia is considered to be a variety of pseudo-leukemia. In the yellow, waxen countenance of its subjects, in its clinical course, which is sometimes remittent or intermittent, in its intensity and its fatality, the disease closely resembles pernicious anemia. Unlike the latter affection, splenic anemia presents a definite visceral lesion, a relative increase of white cells, and a diminution in the relative percentage of hemoglobin.

Chlorosis.

What are the symptoms of chlorosis?

Chlorosis or *green-sickness* is a depraved condition of the blood, seen especially at about the time of puberty in young women, with derangement of menstruation. In addition to the symptoms of simple *anemia*, the *complexion* and the conjunctivæ present a yellowish-green hue. The number of *red corpuscles* is not diminished in the same degree as is the percentage of *hemoglobin*, while the number of *leukocytes* is not appreciably altered.

Pernicious Anemia.

What are the symptoms of pernicious or idiopathic anemia?

There is a form of anemia in which the impoverishment of the blood is marked, and which pursues a progressive and usually fatal course. It is more common in women than in men. No constant visceral lesion has been found associated with the disease. In some instances, there has been atrophy of the gastric glands; in others, disease of the medulla of the bones; in others, increase of iron-containing pigment in the liver; in still others, no lesion except that of the blood has been detected. The disease sometimes appears in connection with pregnancy.

The *symptoms* are those of intense anemia, with irregular outbreaks of febrile temperature. The *complexion* is pallid and often assumes a strikingly yellowish (lemon) hue. The lips and palpebral conjunctivæ may be white. The bones, especially the sternum, exhibit tenderness on pressure. The *pulse* is usually rapid. The *urine* may be notably dark-colored, and contains an excess of nitrogenous matters.

Sometimes deceptive remissions in the symptoms occur. The layer of subcutaneous fat is often well-preserved. Hemorrhages take place from the mucous surfaces, beneath the skin, into the retina and elsewhere, as a result of fatty degeneration of the coats of the arteries. The *heart* is also likely to undergo fatty degeneration.

The volume of *blood* is small; the number of *red corpuscles* is considerably diminished (less than 2,000,000 per c. mm.), as is necessarily the absolute quantity of *hemoglobin*, of which, however, the relative proportion per corpuscle is increased. The red corpuscles are poorly developed and ill-shaped. Megalocytes preponderate; microcytes and poikilocytes are likewise found. In some cases minute, highly-colored globules resembling "small red-tinged, fat globules" are seen. The number of white corpuscles may remain unaltered; in some cases, it has has been increased; in others, diminished.

What special precautions must be taken in arriving at a diagnosis of the various forms of anemia?

When a condition of anemia is discovered, a searching inquiry into the history and symptoms and a careful physical examination must be made, to determine whether or not there be a coëxistent morbid condition, such as carcinoma, tuberculosis, hemorrhoids or other source of hemorrhage, nephritis, malarial infection, intestinal parasites, defective or insufficient nutrition, mal-assimilation, arsenical, plumbic, mercurial or other form of poisoning, and conditions that occasion jaundice.

Leukocytosis.

What is leukocytosis?

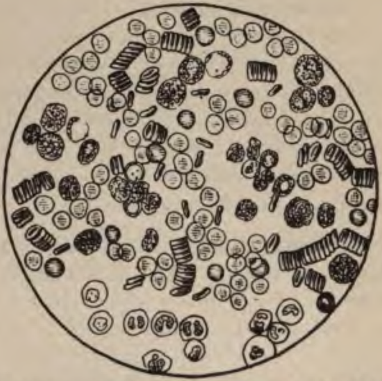
Leukocytosis is a condition of the blood in which, without alteration in the number of red corpuscles, without enlargement of the spleen, of the liver or of the lymphatic glands, the number of white corpuscles undergoes an intermittent or transitory increase. It may be *physiologic*, as when it occurs after meals, or it may be a *transitional* stage of leukemia or pseudo-leukemia.

Leukemia.

What is leukemia or leukocythemia?

Leukemia is a morbid condition in which, in association with enlargement of the spleen, of the liver, of lymphatic glands, or with alterations in the medulla of bones, the blood contains a permanent excess of *leukocytes* and is deficient in *red cells* (Fig. 18).

FIG. 18.



Appearance of the blood in leukemia. (Funke.)

Three varieties of leukemia are recognized: *lienal*, *lymphatic* and *medullary*, as the spleen and liver, the lymphatic system, and the medulla of bones, respectively, are involved.

Early in the course of the disease, the *spleen* becomes conspicuously enlarged; sometimes friction-fremitus may be detected on palpation, and a blowing murmur on auscultation; ascites may develop. In the further course of the disease, the liver and the lymphatic glands may also become enlarged; the bones may be painful and tender; changes in the kidneys and in the intestinal glands are sometimes noted.

The number of *white corpuscles* in the blood is increased beyond 10,000 to the cubic millimeter, sometimes in extraordinary degree. The number of *red cells* and the quantity of *hemoglobin* are diminished. Through increase in the number of the white, and diminution in that of the red, there ensues a great change in the ratio of white cells to red cells. The ratio is, as a rule, not less than 1 : 50, and may reach or exceed 1 : 1. Masses of leukocytes may occasion infarcts in the spleen or lungs. It is said that in lienal leukemia large leukocytes predominate; in lymphatic leukemia, small leukocytes; and in myelogenous leukemia, large leukocytes, having large nuclei, together with transition-types and many eosinophile cells.

The depravity of the blood occasions dyspnea and edema; conjoined with the secondary changes in the bloodvessels, it predisposes to extravasation of the blood. There are thus breathlessness on exertion, epistaxis and other hemorrhages from mucous surfaces and into the retina and subcutaneous connective tissue. Diarrhea is common. The *urine* contains an excess of uric acid. Rarely, disturbances of vision occur—as a result of hemorrhage, of leukemic deposit, or of leukemic retinitis. From time to time febrile periods, lasting from a few days to a week, in which the temperature may rise to 102° F., are observed. Remissions in all of the symptoms sometimes take place during the progress of the case.

Leukemia is most common between the ages of twenty and fifty; it is more common in men than in women. It is usually insidious in *onset*, the early clinical phenomena being those of anemia in general. Epistaxis is often the first significant symptom. The *etiology* of leukemia is obscure. In some cases there has been a history of antecedent malarial infection; in others, of traumatism of the spleen; in others, of traumatism of bones. Privation and exposure are cited among predisposing and exciting causes.

Pseudo-leukemia.

What is pseudo-leukemia?

Pseudo-leukemia, *Hodgkin's disease*, *lymphatic anemia*, *malignant lymphoma* or *lymphadenoma*, is an affection characterized by hyperplasia of adenoid tissues, and especially by enlargement of the lymphatic glands in various parts of the body, and by changes in the blood. *Anemia* prevails, the number of *red corpuscles* and the relative proportion of *hemoglobin* being diminished, the number of *white corpuscles* remaining unchanged or not uncommonly being slightly increased. An apparent excess of white cells, due to decrease in the number of red cells, with consequent moderate elevation of the ratio, must not be mistaken for an actual increase in the number of leukocytes. The superficial glands, especially those of the neck and axilla, are usually chiefly involved. Sometimes the glandular enlargement is first noticed in the groin. The spleen and other viscera may also be enlarged. In addition to the symptoms of anemia and malnutrition, pressure-phenomena are observed. Enlargement of the tonsils, of the lingual, postnasal, cervical, peri-tracheal and peri-bronchial glands gives rise to cough, dyspnea and dysphagia. The cerebral circulation may be impeded from pressure upon the vessels of the neck. Pressure upon nerves may cause paralysis, pain or edema. Jaundice and ascites are sometimes observed. There are, in addition, a tendency to effusions, hemorrhages and petechiæ, and from time to time febrile periods.

How are leukemia and pseudo-leukemia to be differentiated?

It is possible that leukemia and pseudo-leukemia represent stages of a single condition. Individual cases present symptoms of both affections. The differentiation of well-marked cases, however, is not difficult.

In leukemia, the spleen enlarges early; the lymph-glands are not necessarily involved; when they are, however, it is late in the disease. In pseudo-leukemia, the lymph-glands are primarily enlarged; the spleen and liver may escape entirely; should they become involved, it is late in the disease. In leukemia, the number of white blood-cells is always increased—often in decided degree. The alteration of relation of white cells to red cells in pseudo-leukemia is due to decrease in the number of the red cells; actual excess of white cells constitutes no essential part of the disease; though not rarely observed, it never reaches the high figures of leukemia.

How are tuberculosis of the lymph-glands ("scrofula") and pseudo-leukemia to be differentiated?

The lymphatic glands over a wide distribution may become tuberculous. Such a condition will also be associated with anemia—so that a clinical picture will be presented simulating that of pseudo-leukemia. Microscopic examination of a portion of one of the growths will disclose the presence of tubercle-bacilli in the one instance and a condition of simple hyperplasia in the other.

In pseudo-leukemia the enlarged glands are not inflamed, and do not give rise to inflammation in the surrounding connective tissue. They are not hot or tender to the touch; they are not adherent to the skin, and are, as a rule, freely movable upon each other. Tuberculous glands are usually adherent to the skin, and to each other, and manifest a tendency to chronic inflammation, with caseation and suppuration. There are usually other evidences of tuberculosis present, in the bones, joints, or elsewhere; and the *facies* is often significant. Glandular tuberculosis and pseudo-leukemia may, however, coëxist; or inflammatory changes may be set up by traumatism in the glands of pseudo-leukemia.

Infantile Pseudo-leukemic Anemia.

What is infantile pseudo-leukemic anemia?

Jaksch has described in infants a form of anemia in the last stage of which there are a deficiency of *red corpuscles*, a deficiency of *hemoglobin*, and an increased number of *white corpuscles*.

The condition differs from leukemia in that the increase of white corpuscles is not so great, that eosinophile leukocytes are never present, that, while the spleen is enlarged, the liver is not, and that the prognosis is favorable.

Table showing alterations in the constitution of the blood in disease.

	Number of red cells.	Number of white cells.	Proportion of hemoglobin.	
			Total.	Per red cell.
Anemia	Diminished.	Normal or diminished.	Diminished.	Normal or diminished.
Chlorosis	Diminished.	Normal or diminished.	Diminished.	Diminished.
Pernicious anemia	Greatly diminished.	Variable.	Greatly diminished.	Increased.
Splenic anemia	Diminished.	Increased.	Diminished.	Diminished.
Pseudo-leukemia	Diminished.	Normal or increased.	Diminished.	Normal or diminished.
Leukocytosis	Normal.	Increased.	Normal.	Normal.
Leukemia	Diminished.	Greatly increased.	Diminished.	Normal or diminished.

Scorbutus.

What are the symptoms of scorbutus?

Scorbutus or *scurvy* is a disease dependent upon deficiency in the diet, of certain substances usually contained in the juices of fresh vegetables and fruits. It was formerly common at sea, and in jails and poorhouses. Outbreaks still occasionally occur. Isolated cases sometimes appear under unexpected circumstances.

The disease is characterized by a depraved condition of the blood, with degeneration of the walls of the vessels and consequent hemorrhages. The *gums* are soft and spongy, and bleed on slight provocation or spontaneously. The *breath* is defiled by the fetor of the decomposing necrotic tissue. Hemorrhages take place from other mucous surfaces, while inflammation of serous membranes, with hemorrhagic effusions, may occur. Extravasations of blood take place into the subcutaneous tissues, especially in the course of the superficial veins of the dependent parts of the body, giving a mottled, bluish or purplish appearance to the skin in more or less extensive areas. The discoloration frequently resembles that of a bruise, and, as it slowly fades, assumes a greenish tint. It is often quite persistent. Extravasation into the muscles may likewise occur. With this, edema is often associated. Brawny induration of the connective tissue in various parts of the body may develop. Diffuse, dull pains are felt. The *skin* is dry and rough. There is mental apathy, with a sense of lassitude and an undue readiness of fatigue. Fungated hemorrhagic ulcers form. Existing ulcers assume an unhealthy, spongy appearance. Fractured bones fail to unite, while the union of broken bones may be dissolved. The *complexion* is sallow ; anemia becomes evident, with shortness of breath and rapidity and feebleness of pulse. In some epidemics, dysentery has been a *complication*. Sometimes hemeralopia or night-blindness has been observed. Without proper treatment death takes place. With proper treatment recovery is slow.

Purpura.

What are the clinical features of purpura?

Purpura is a manifestation of a deteriorated condition of the blood and vessels, as a result of which petechiæ form, and hemorrhages from the mucous membranes take place. Sometimes blood is extravasated into the serous cavities ; and occasionally intracranial hemorrhage occurs. The disease sometimes develops in the midst of apparent health ; at other times, in the course of nephritis, the exanthemata, hepatic disease, and rheumatism. Occasionally, transient febrile periods are observed. Sometimes the disease *begins* acutely, with a chill followed by pain in the back or limbs, but without fever. The purpuric spots may be large or small, of limited or of extensive distribution. Usually they appear in successive crops. At first reddish, they soon become of a deep purple, gradually fading to brown, and then to yellow, as they disappear.

The varieties of purpura commonly recognized are *purpura simplex*, *purpura hemorrhagica* and *purpura rheumatica*. The names are sufficiently descriptive.

How are scorbutus and purpura to be differentiated?

Scorbutus occurs among those that are massed together and are insufficiently supplied with fresh vegetables; purpura may develop amid circumstances apparently the most favorable and in the face of an abundant supply of fruits and vegetables. The gums may bleed in purpura, but they are wanting in the sponginess and lividity of scorbutus. The breath is fetid in scurvy, not necessarily so in purpura. The petechiæ of scorbutus usually develop about the hair-follicles; those of purpura are indifferent in distribution. The individual discolored patches are more extensive in scorbutus than in purpura. In the latter, large blotches can usually be resolved into a number of smaller spots aggregated. The hue in the two cases differs to the experienced eye.

How is purpura to be distinguished from measles?

The eruption of purpura has in certain instances presented sufficient superficial resemblance to that of morbilli to occasion mistake—the youth of the patients and the coincidence of febrile symptoms increasing the difficulty. The course of the eruption is, however, quite different in the two affections. The distribution of the purpuric spots is not so general as is that of the eruption of measles. The spots of purpura often appear first on the legs, while in measles the rash begins on the face and trunk. The purpuric spots change color and fade, new spots appearing while others are receding; no such phenomenon occurs in measles. Catarrhal symptoms do not occur in purpura; hemorrhages are not usual in measles. The characteristic temperature-curve of measles is absent from the course of purpura.

Hemophilia.

What are the clinical features of hemophilia?

Hemophilia is a morbid condition manifested by an abnormal tendency to the occurrence of hemorrhages, spontaneously or upon slight provocation. Individuals so affected are called "bleeders." The disease occurs in families; it is much the more common in males, but is mostly transmitted through females, in whom, when it occurs, its manifestations are always mild. Slight wounds, a scratch, the extraction of a tooth, may be followed by alarming or even fatal hemorrhage. Petechiæ form, while sometimes large extravasations of blood take place into the subcutaneous textures and into the joints. The disease usually first makes its appearance early in life. The *diagnosis* depends essentially upon the history of hereditary transmission, the unusual proneness to the occurrence of alarming hemorrhage, the formation of petechiæ, the development of subcutaneous and articular extravasations of blood, and the appearance of the first symptoms early in life.

Addison's Disease.

What are the clinical features of Addison's disease?

Addison observed that in certain cases, in which after death the *supra-renal bodies* were found tuberculous, there had existed during life a peculiar pigmentation of the skin, with remarkable asthenia and nausea and vomiting. The discoloration is usually of a brownish hue, like that which develops in one exposed to a tropical sun; it resembles the pigmentation of the dark-skinned races. It occurs in plaques, on exposed surfaces, at parts that have been compressed or constricted, in the flexures of joints, and about the genitalia and the nipples. The mucous membrane of the mouth and tongue may be pigmented. Progressive weakness is manifested; and prostration finally develops. The subcutaneous fat, however, may be preserved. The *heart's action* is feeble; the *pulse* is small and compressible. The *appetite* is impaired; nausea and vomiting are common; there may be diarrhea or constipation. *Death* may take place gradually, from exhaustion, or with unexpected suddenness.

Tuberculous disease of the supra-renal bodies has been found when there was no bronzing of the skin. In such cases it has been thought that the disease had not progressed sufficiently to have occasioned symptoms. In other cases the skin has been pigmented, but no changes were found in the supra-renal bodies. Pigmentation of the skin may, however, be a result of other conditions than supra-renal disease. By some, Addison's disease is thought to depend upon changes in the semilunar ganglia and branches of the sympathetic system of nerves.

Rachitis.

What are the clinical features of rachitis?

Rachitis is a disease dependent upon defective and perverted development of the osseous structures of growing children, probably as a consequence of faulty nutrition. There is doubtless congenital predisposition to its occurrence. The *bones* are soft and yielding, and wanting in their natural firmness and stability, so that various deformities result. The sides of the chest become flattened, and the sternum projects, giving rise to the "pigeon-breast." Nodules or "beads" form at the junction ot the ribs and their cartilages. The long, supporting bones become bent and their epiphyseal extremities enlarged. The soft and deformed bones are especially liable to green-stick fractures. The *lower jaw* is narrow and dentition is delayed. The teeth may decay and fall out soon after their appearance. The *head* appears large; its summit is flat; the fontanels close late. The deformities are maintained by the ultimate hardening of the affected bones.

Rachitic children present a pasty complexion and pearly conjunctivæ; they are undersized and poorly resist disease. They are restless, and display a tendency to excessive sweating of the head. *Digestion* is impaired; the *abdomen* is often protuberant. Laryngismus stridulus and convulsions are common occurrences. Internal viscera may be enlarged as a result of hyperplasia of the interstitial connective tissue.

Mollities Ossium.

What are the clinical manifestations of mollities ossium?

Mollities ossium is a morbid condition that develops in adults amid unfavorable hygienic surroundings and in women that have borne many children. It manifests itself by both softening and rarefaction of the bones, so that progression is impossible, and fractures are common. Occasionally febrile symptoms are present. *Death* is the common result, either from exhaustion, or from mechanical interference with respiration.

How are rachitis and mollities ossium to be differentiated?

Mollities ossium is a disease of adult life, attended by changes in developed bone, and usually of fatal termination. Rachitis is essentially a disease of childhood, dependent upon abnormalities in developing bone, and from which recovery usually takes place, only the sequels of the disease remaining.

THE HEART.

Inspection.

What can be learned of the heart by inspection?

On *inspecting* the *normal chest*, one perceives in the left fifth intercostal space, two inches below and an inch within the nipple, in an area of perhaps an inch in diameter, a gentle rise and fall—the *cardiac impulse* or *apex-beat.* It is less distinctly visible in fat than in lean persons.

The position of the apex-beat varies slightly with the respiratory movements, with posture, and with the state of the abdominal viscera. It may be *displaced*, as to the left by an effusion in the right pleural cavity, or by adhesions of the left pleura; or to the right by an effusion in the left pleural cavity, or by adhesions of the right pleura. It may be *increased* in extent, as when the heart is enlarged, or when the pericardium is distended with fluid. Under varying conditions an impulse is seen in the epigastrium.

The impulse may be *strong*, as in cases of cardiac hypertrophy; *feeble*, as in cases of dilatation; *wavy*, as when the pericardial cavity is occupied by fluid; when the pericardium is adherent "systolic dimpling" occurs.

Palpation.

What is to be learned of the heart by palpation?

Palpation confirms and reinforces what is learned by inspection. An impulse that cannot be seen can sometimes be felt. A *feeble* impulse indicates that the action of the heart has been enfeebled by disease or by an effusion in the pericardial cavity; a *strong* impulse is indicative of strength, of hypertrophy. A pericardial friction-rub may sometimes be felt. In most cases of mitral valvular obstruction, a *purring tremor* is perceived on palpation.

Percussion.

What is to be learned of the heart by percussion?

By *percussion*—which is best practised with the patient recumbent—the approximate *size* of the heart can be learned. A considerable portion of the organ is covered by lung-tissue. By *superficial percussion* of a normal chest the cardiac dulness is found to be represented by a triangle included between a point at the lower margin of the fourth left costal cartilage at its junction with the sternum, another at the apex-beat, and a third at the lower extremity of the sternum at its left border. *Deep percussion* defines a somewhat more extended area.

The area of cardiac percussion-dulness is *increased* when the heart is enlarged, or the pericardial sac is distended by fluid, when the heart is uncovered by retraction of the lung, or in full expiration. The area is *diminished* when the heart is covered by emphysematous lung-tissue, or by the lungs in full inspiration.

Auscultation.

What is to be learned of the heart by auscultation?

Auscultation constitutes the most important method of physical examination of the heart. It reveals the frequency, the rhythm, the quality, and the purity of the heart-sounds.

In health, the heart of an adult at rest beats about seventy-two times to the minute. The *frequency* of action varies with the degree of bodily exertion, and with posture. It is *increased* by exertion and by excitement, after meals, in febrile affections and in many diseases of the heart; it is greater in the upright than in the horizontal posture; it is *diminished* by rest and by jaundice; in aortic obstruction in fatty degeneration, and during convalescence from acute disease. It is affected by various drugs and by nervous influences.

The action of the heart in health is *rhythmical* and *regular*. There is a prolonged, dull, *first sound*, and a shorter, sharper *second sound*, followed by an *interval of silence*—with rhythmical repetitions. Occasionally a cycle is omitted—the heart *intermits*. A normal first sound may be followed by two second sounds—*duplication*. The first sound may also be duplicated. The normal rhythm of the heart may be disturbed by organic disease of the heart, such as degeneration, and by derangement of other organs, as of the stomach.

The *first sound* of the normal adult heart is a dull, but well-defined thud; the *second* is a shorter, sharper, snapping or ringing sound; it is followed by an *interval of silence*.[1]

Three elements enter into the production of the first sound, which is synchronous with the contraction of the ventricles (*systole*) and the closure of the auriculo-ventricular (*mitral* and *tricuspid*) valves. These are: the *closure of the valves*, the *muscular contraction*, and the *impact of the heart* against the chest-wall. The second sound, which is synchronous with the beginning of the *diastole*, is valvular; it is due to the quick approximation of the semilunar flaps, preventing return of blood through the arterial (*aortic* and *pulmonary*) orifices.

The first sound of the heart may be *altered* in volume, in tone, in duration and in strength. These are usually *increased* when the heart is hypertrophied, temporarily under excitement and after the administration of certain drugs; and *diminished* when from any cause the action of the heart is enfeebled—among such causes are dilatation, degeneration, pericardial effusion. The character of the second sound is largely dependent upon the tension in the arteries; the higher the tension the sharper the sound.

---

[1] There can likewise be detected by the trained ear a short interval of silence between the first and second sounds. The fact is mentioned here to avoid misleading the student in his further studies; but for practical purposes this "minor silence," as it is called, may be entirely ignored.

The *purity* of the heart-sounds depends upon the condition of the heart-muscle, upon the state of the blood and upon the functional efficiency of the various valves and orifices.

The functional *efficiency* of the valves and orifices of the heart can be determined by a study of the sounds of the heart as heard over the respective parts; but these are so close together that points in the course of the blood-stream are selected for auscultation. That for the mitral sound corresponds to the situation of the apex-beat; that for the aortic, to the junction of the right second costal cartilage with the sternum; that for the tricuspid, to the ensiform cartilage; and that for the pulmonary, to the left second intercostal space close to the sternum.

Alteration of the structure and derangement of the function of the valves and orifices of the heart are revealed by adventitious sounds, called *murmurs*, that accompany or replace the normal sounds, or occur in the interval between them. Murmurs are often blowing; sometimes they are soft, sometimes harsh, sometimes musical.

The first sound occupies the ventricular *systole;* the *diastole* is taken up by the second sound and the period of *silence*. During the systole, the blood pases from the ventricles into the aorta and pulmonary artery; the arterial valves should be freely open; the auriculo-ventricular valves should be perfectly closed: murmurs generated at the arterial orifices indicate obstruction to the outflow of blood from the ventricles; murmurs generated at the auriculo-ventricular orifices indicate reflux of blood into the auricles.

At the conclusion of the systole, there is a brief pause, followed by the diastole, during which the blood flows from the auricles into the ventricles; the auriculo-ventricular valves should be freely open; the arterial valves should be perfectly closed: murmurs generated at the auriculo-ventricular orifices indicate obstruction to the onflow of blood into the ventricles;

murmurs generated at the arterial orifices indicate reflux of blood from the arteries.

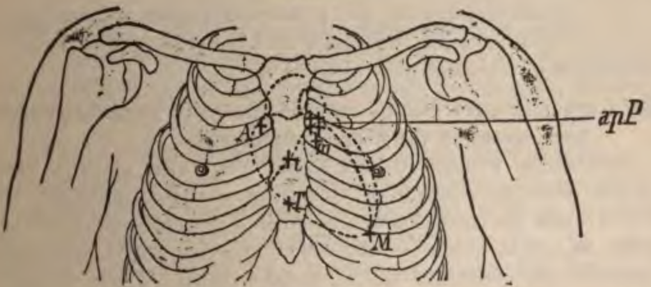
Incompetency or insufficiency of a valve permits of regurgitation.

Constriction or occlusion of an orifice occasions obstruction.

The seat and character of an endocardial murmur are determined from the *place* at which it is best heard (site of *maximum intensity*), in association with the *time* of its occurrence and the direction in which it is *transmitted*.

Analogous conditions of the right and left heart necessarily give rise to murmurs at the same time; analogous conditions of the auriculo-ventricular and arterial valves give rise to murmurs at different times; and conversely. Thus, for example, the murmurs of mitral insufficiency and of tricuspid insufficiency coincide in time with each other and with the murmurs of aortic obstruction and of pulmonary obstruction. Mitral and tricuspid regurgitant murmurs and aortic and pulmonary obstructive murmurs are heard during the systole—with or in place of the first sound. Mitral and tricuspid obtructive murmurs and aortic and pulmonary regurgitant murmurs are heard during the diastole; the latter two with or in place of the second sound;

FIG. 19.



Auscultation of the heart-sounds. The small letters indicate the situation of the valves; the large letters, the points for *auscultation*. *aA*, aortic; *mM*, mitral; *tT*, tricuspid; *pP*, pulmonary. (Vierordt.)

the first two, however, in what is normally the period of silence immediately preceding the first sound—hence *presystolic.*

Mitral regurgitant murmurs are heard best at the apex of the heart; they are transmitted in the course of the fifth and sixth ribs to the axilla, and may be heard below the posterior inferior angle of the scapula. *Mitral obstructive* murmurs are best heard at the apex of the heart, or a little above the apex; they are but feebly transmitted. *Aortic regurgitant* and *aortic obstructive* murmurs are most distinctly heard at the junction of the second costal cartilage on the right with the sternum; the former are transmitted downwards in the course of the sternum, the latter upwards in the course of the great vessels, especially the carotid. Murmurs generated at the orifices and by the valves of the right side of the heart are exceedingly rare. *Tricuspid regurgitant and obstructive* murmurs are best heard at the ensiform cartilage, or a little farther to the right. *Pulmonary* murmurs should be best heard in the second intercostal space on the left, close to the margin of the sternum. In anemia, soft blowing murmurs, dependent upon the condition of the blood, are heard in the same situation.

All murmurs heard in the precordial region are not endocardial. Auscultation reveals as well the sounds of pericardial friction as of adjacent pleural friction.

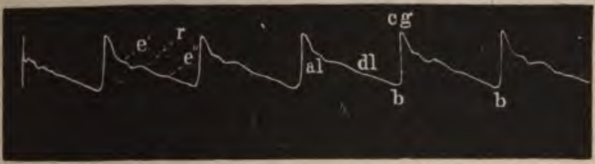
How are murmurs due to organic disease to be distinguished from so-called functional murmurs?

In addition to adventitious sounds generated at the orifices of the heart as a result of structural changes, murmurs are heard when the condition of the blood is deteriorated, or when, from disturbed action of the heart or other cause, abnormal currents are generated in the blood-stream. These so-called *functional murmurs* are distinguished by their inconstancy and their softness; they are usually heard only at the base of the heart and over the body of the organ; they are not transmitted; they are intensified by pressure with the stethoscope; and they disappear with the removal of the conditions upon which they depend. Organic murmurs are usually harsher, more constant, and vary comparatively little in character and intensity.

What is the sphygmograph?

The sphygmograph is an instrument by which an artery is made to record certain of the characters of its pulsation. The *sphygmogram* (Fig. 20) is an important aid in diagnosis, but cannot be relied upon apart from the ordinary rational and physical signs. The use of the sphygmograph and the significance of its tracings must be learned by experience.

Fig. 20.



Normal pulse-tracing. (After Eichhorst.)

Malformation.

What are the most common malformations of the heart?

The most common malformations of the heart consist in an imperforate interventricular septum and a failure of the foramen ovale to close.

To what symptoms do malformations of the heart give rise?

Individuals in whom there exist serious abnormal communications between the lateral halves of the heart rarely reach adult life. Cyanosis is the most common symptom. Systolic murmurs are heard practically indistinguishable from those occasioned by valvular derangement.

Dextrocardia.

What is dextrocardia?

Dextrocardia is a congenital displacement of the heart on the right side, commonly associated with displacement of the liver on the left and of the spleen on the right.

How is dextrocardia to be recognized?

In case of dextrocardia the impulse of the heart is wanting in its usual situation, and is seen to the right of the sternum. The area of cardiac percussion-dulness occupies on the right an extent corresponding to that which it normally occupies on the left. The sounds of the heart are heard on the right side instead of on the left. The hepatic percussion-dulness is not found in its usual situation, but in a corresponding position on the left. The splenic dulness is found on the right instead of on the left.

Functional Disturbance of the Heart.

What is meant by functional disturbance of the heart?

Under various conditions, as when the nutrition is impaired or the digestion is deranged, as a result of overwork, or of dissipation, or of the excessive use of tobacco, or tea, or coffee, and in connection with gout or lithemia, with hysteria or hypochondriasis, the action of the heart may be deranged without recognizable structural change. There will be present such symptoms as pain, palpitation, anxiety, headache, vertigo and breathlessness, sometimes with irregularity and increased frequency of the heart's action. The diagnosis depends upon the recognition of the primary condition and upon the absence of the physical signs of a cardiac lesion. Functional disorder, great in degree and long continued, may lead to structural change.

Tachycardia.

What is tachycardia?

Tachycardia is a term applied to a somewhat rare condition of excessive rapidity of the action of the heart, accompanied with palpitation, the rhythm of the heart sometimes remaining unaffected. The pulse may exceed 200 beats a minute. Occurring in paroxysms, the qualification *paroxysmal* is applied. When no etiologic lesion is discoverable, the condition is termed *essential paroxysmal tachycardia*. The paroxysm usually begins suddenly, with or without warning; at times without apparent exciting cause; at other times seeming to result from some such condition as overdistention of the stomach. Tachycardia may be *due* to temporary paralysis of the vagus or stimulation of the cardiac accelerator nerve. Increased cardiac dulness and indefinite murmurs may be observed during the attack, and disappear with subsidence of the symptoms. The condition may be unattended with other symptoms, and ordinarily does not shorten life. It may be a part of other neuroses. It has been observed in women at the menopause.

How does tachycardia differ from angina pectoris?

Tachycardia is wanting in the threatening symptoms of angina pectoris—the anxiety, the pain, the cardiac failure. Rapidity of the heart's action and palpitation are the essential features of tachycardia; while in angina pectoris the pulse is of variable frequency.

How does tachycardia differ from exophthalmic goiter?

Palpitation of the heart and increased frequency of the pulse are among the earliest phenomena of exophthalmic goiter. Before the thyroid gland has become enlarged or the eyeballs protrude, the distinction from tachycardia is not possible. There is no difficulty in the diagnosis, however, when not only the exophthalmos and the goiter, but the array of other symptoms that characterize exophthalmic goiter, have also developed.

Irritable Heart.

What are the phenomena of irritable heart?

Irritable heart is a condition originally observed in soldiers in active service, in which there are in addition to increased frequency of the action of the heart, often with disturbed rhythm, recurring attacks of palpitation and pain in the precordia. There are usually headache and vertigo, especially during the paroxysms. The general health may suffer little or not at all. The first sound may be short and sharp or may be barely audible; the second sound is accentuated. There is no constant murmur. The pulse is compressible and easily influenced by position. Respiration is but little if at all accelerated. A similar condition may develop in civil life in those unaccustomed to arduous labor called upon to perform unusual tasks. It has also been found in athletes and others who have committed excesses in physical exertion, and in masturbators. Under proper regimen, restoration to the normal results; under other circumstances, hypertrophy of the heart develops.

How does irritable heart differ from tachycardia?

Irritable heart results from well-recognized causes that are not concerned in tachycardia. In irritable heart the frequency is less than in tachycardia, and is habitual; in tachycardia the increased frequency is extraordinary and usually occurs in paroxysms. It is pain and palpitation rather than increased frequency of action that marks the paroxysmal seizures of irritable heart. Tachycardia is also wanting in the distressing subjective sensations and the grave issue of irritable heart.

Angina Pectoris.

What are the characteristics of angina pectoris?

Angina pectoris is a paroxysmal disorder for which no definite structural lesion has been found. Perhaps the most common condition associated with it is atheroma of the coronary arteries.

The attack *sets in* suddenly, with a sense of oppression, dyspnea, and pain in the precordia, rising to a high pitch of intensity, and attended with a sense of impending dissolution—and not uncommonly death does occur in the paroxysm. There is often a sensation as of throttling. The pain in the heart is described as "clutching," "squeezing," and "tearing." The pain radiates in various directions from the heart, especially to the left shoulder, and extends down the left arm. The *face* is pale, the features drawn, the *pulse* variable. The attacks may occur spontaneously, but are usually brought on by excitement or exertion, or by gastro-intestinal derangement. They recur with varying frequency, sometimes over a long period of years.

How does intercostal neuralgia differ from angina pectoris?

The pain of intercostal neuralgia never attains the intensity or presents the peculiar character of that of angina pectoris; nor are the general manifestations ever so portentous. The tender points of Valleix, found in intercostal neuralgia, are absent in angina pectoris. An herpetic eruption on the chest, following the course of an intercostal nerve, is diagnostic of neuralgia.

Hypertrophy.

What are the symptoms of hypertrophy of the heart?

The size of the heart is proportionate to the demands made upon it, or to the stimulation that it receives. Valvular disease and other conditions impeding the circulation may cause one or all of the cardiac chambers to enlarge and the walls to thicken. Hypertrophy, uncomplicated by valvular disease, results when the heart is called upon to perform excessive labor, or is stimulated by abnormal nervous influences. Symptoms arise only when the action of the heart is in excess of the requirements of the system. Under such conditions there will be a sense of discomfort in the precordial region, palpitation, paroxysmal cough, shortness of breath, headache, vertigo, ringing in the ears, disturbed sleep, deranged digestion, a florid complexion, and a tendency to hemorrhage.

What are the physical signs of hypertrophy of the heart?

When the heart is *hypertrophied*, its *impulse* is decided and extended, and usually displaced to the left. The area of *percussion-dulness* is increased. As the enlargement, in most cases, principally involves the left ventricle, the area of dulness increases to the left, though enlargement of the left ventricle is likely to be followed in turn by hypertrophy of the remaining cavities. Owing to the position of the heart in the chest, the left ventricle may be moderately enlarged without giving rise to appreciable percutory abnormality. Enlargement of the right heart may be the first cause of extension of the area of dulness. The *action* of the heart is moderately accelerated, regular and rhythmical. The *first sound* is strong and booming, the *second* accentuated. The *pulse* is correspondingly full and strong.

Dilatation.

What are the symptoms of dilatation of the heart?

Dilatation of the heart occurs under pretty much the same conditions as give rise to hypertrophy, except that the organ is unable to meet the demands made upon it. Dilatation may thus be a sequel of hypertrophy. As dilatation is frequently an ultimate result of obstruction to the pulmonary circulation, the right heart usually suffers the more. The symptoms are those of *failing circulation:* precordial anxiety, palpitation, headache, vertigo, syncope, pallor, cough, dyspnea, venous congestion, and dropsy.

What are the physical signs of dilatation of the heart?

The cardiac *impulse* is feeble and diffused, and usually displaced to the left. The area of cardiac *percussion-dulness* is increased. The *first sound* is weakened in correspondence with the disproportion between the enlargement of the cardiac chambers and the thickness of their walls; the *second* is little changed. If the dilatation has been great enough to so enlarge the orifices or weaken the muscles that the valves are no longer competent to effect complete closure, regurgitant murmurs may be developed, even in the absence of structural alterations in the valves. Such murmurs are usually rather soft and may be inconstant. The *action* of the heart may be rapid and irregular. The *pulse* is small and soft.

What are the distinctions between hypertrophy and dilatation of the heart?

HYPERTROPHY.	DILATATION.
Face florid; ringing in ears; rushing of blood to the head.	Face pallid; tendency to syncope; dropsy.
Cardiac impulse strong, extensive.	Impulse feeble, diffused, often wavy.
Increased area of percussion-dulness.	Increased area of percussion-dulness.
Action rapid, regular, rhythmical.	Action rapid, irregular.
First sound strong.	First sound relatively enfeebled.
Pulse full, strong.	Pulse small, yielding.

How does dilatation of the heart differ from fatty degeneration of the heart?

Fatty degeneration of the heart may be recognized when the skin has a peculiar, greasy appearance, when an arcus senilis is present, when the sounds of a heart not enlarged are exceedingly feeble, and attended with the symptoms of a failing circulation. It differs from dilatation in the unchanged or diminished size of the heart and the association with such other evidences of fatty degeneration as may be present. The pulse is usually, but not invariably, slow in fatty degeneration, while it is rapid in dilatation.

How is a pericardial effusion to be distinguished from dilatation of the heart?

Fluid may collect in the pericardium in connection with pericarditis, or as a part of a general dropsy. In dilatation of the heart the cardiac impulse is feeble and diffuse, but not as feeble or as wavy as in effusion. When dilatation exists, the percussion-dulness in the precordia is not as extensive as when there is an effusion, nor is it peculiarly triangular in outline. While the sounds of the heart are enfeebled over the organ when dilatation exists, they are almost obliterated in case of effusion, except at the base, where, in case of pericarditis, friction-sounds may likewise be detected. The recognition of a condition that gives rise to pericardial effusion is an aid in diagnosis.

Valvular Disease.

What are the symptoms common to valvular lesions of the heart?

The functional deficiency of a diseased heart-valve may be compensated for by an improved condition of the heart-muscle. The presence or absence of rational signs will depend upon the extent and constancy of the structural and functional alteration of the muscle. If the alteration be too great, the symptoms will be those of an hypertrophied or overacting heart. If it be too little, the symptoms will be those of an enfeebled heart. If it be inconstant, symptoms of excitement, of overaction or of enfeeblement may appear in paroxysms, or for prolonged periods at irregular intervals. Even when the compensatory change is apparently constant and sufficient, there may be at times or continuously, with or without pallor and weakness, a sense of precordial discomfort, palpitation of the heart, shortness of breath, perhaps headache and vertigo—all aggravated by exertion. Often, when compensation is but slightly imperfect, there are evidences of gastric and intestinal derangement, and a sense of fulness in the hypochondria. Should the heart fail, and compensation be disturbed, the condition becomes virtually or actually that of dilatation of the heart; the phenomena are those of insufficient *vis a tergo* in the circulation. As a result of the stagnation of blood in the veins, there is engorgement of various organs, especially of the lungs, liver, kidneys and spleen; the existing symptoms become intensified, and in addition there appears dropsy, first manifesting itself in the lower extremities, and extending upwards.

When two or more lesions coëxist, the compensation is less readily established and more easily ruptured than in the case of single lesions. Pulmonary congestion and hemoptysis occur not infrequently.

Mitral Incompetency—Mitral Regurgitation.

What are the signs of incompetency of the mitral valve (mitral regurgitation)?

As a result of endocarditis, or of fibrous or calcareous degeneration, the bicuspid leaflets, or the chordæ tendinea, become thickened and contracted, interfering with the accurate apposition of the segments of the mitral valve during the systole. In consequence, blood is abnormally diverted backward through the mitral orifice, giving rise to a blowing (systolic) sound, heard with greatest distinctness at the apex, and transmitted, in the course of the fifth and sixth ribs, to the axilla and to the inferior angle of the scapula. The heart is usually enlarged—under favorable conditions, hypertrophied; under unfavorable conditions, dilated.

Mitral Obstruction.

What are the physical signs of obstruction at the mitral orifice?

The mitral orifice becomes contracted as a result of endocarditis or of degenerative changes in the segments of the valve. The characteristics of mitral obstruction are: a rumbling murmur heard just before the systole, with greatest intensity over the left ventricle, and a purring tremor or thrill perceived by the hand placed over the precordia. The left ventricle diminishes in size, while the left auricle becomes enormously enlarged. In turn the right side of the heart becomes enlarged. The pulse is small and feeble. Not rarely mitral incompetency is associated with obstruction.

Aortic Obstruction.

What are the physical signs of aortic obstruction?

Aortic obstruction is a result of structural changes in the semilunar valves or in the aorta, induced by inflammation or degeneration. Aortic obstruction and incompetency are often associated. The action of the heart is strong; the left ventricle becomes hypertrophied; the pulse may be full and strong or small and tardy. At midsternum and on the right side over the junction of the second costal cartilage with the sternum, a coarse, blowing, systolic murmur is heard, transmitted into the great vessels. The murmur of aortic obstruction is sometimes to be heard over the right carotid artery when at midsternum and at the aortic cartilage, merely an obscuration of the first sound can be detected.

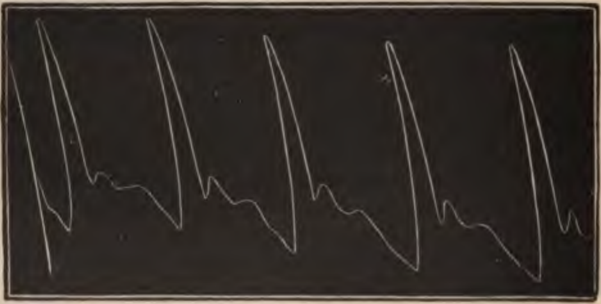
Aortic obstruction, apart from insufficiency, is one of the less common lesions. A systolic murmur heard at the aortic cartilage and transmitted into the neck may be due to dilatation or roughening of the aorta, without valvular or orificial change, and may give rise to no circulatory disturbance.

Aortic Incompetency—Aortic Regurgitation.

What are the physical signs of incompetency of the aortic valve (aortic regurgitation)?

Aortic incompetency arises from conditions similar to those that give rise to imperfections at the other orifices of the heart. It is often associated with aortic obstruction. It is characterized by a blowing, diastolic murmur, replacing or accompanying the second sound of the heart, heard with greatest intensity at the aortic cartilage, and transmitted downwards in the course of the sternum. The action of the heart is powerful; the left ventricle becomes enormously hypertrophied; vascular pulsation is common and may be evident even in parts remote from the center of circulation; the pulsation in the carotids may be so strong that the head is shaken; the pulse comes up well with the systole of the heart, but immediately recedes—constituting the gaseous pulse, the water-hammer pulse, or the pulse of Corrigan. This character is well shown in the sphygmographic tracing. (Fig. 21.)

Fig. 21.



Pulse-tracing of aortic insufficiency. (After Strümpell.)

Tricuspid Incompetency—Tricuspid Regurgitation.

What are the signs of tricuspid regurgitation (incompetency of the tricuspid valve)?

Lesions of the valves and orifices of the right side of the heart are uncommon.

If the tricuspid valve is incompetent, blood regurgitates with the contraction of the ventricle; in consequence, a blowing systolic murmur is heard at the ensiform cartilage, and systolic pulsation is visible in the veins of the neck. In some cases the liver is seen to pulsate.

Tricuspid Obstruction.

What are the signs of obstruction at the tricuspid orifice?

When the tricuspid orifice is obstructed the blood must accumulate in the peripheral veins; a presystolic murmur is heard at the ensiform cartilage.

Pulmonary Obstruction.

What are the signs of obstruction at the pulmonary orifice?

When the pulmonary orifice is obstructed a systolic murmur is heard in the third intercostal space on the left, close to the sternum.

Pulmonary Incompetency—Pulmonary Regurgitation.

What are the signs of incompetency of the pulmonary valve (pulmonary regurgitation)?

When the pulmonary valve is incompetent regurgitation occurs with the dilatation of the ventricle; as a result a diastolic murmur is heard at the junction of the third costal cartilage on the left with the sternum; the murmur may be transmitted downwards in the course of the sternum.

Endocardial Murmurs.

Time of occurrence.	Site of greatest intensity.	Direction of Transmission.	Seat of lesion.	Nature of lesion.
Systolic.	At cardiac apex.	Along left fifth and sixth ribs—in left axilla—in the back, at inferior angle of left scapula.	Mitral orifice.	Incompetency—Regurgitation.
Systolic.	At junction of right second costal cartilage with sternum.	To junction of right clavicle with sternum—in course of right carotid.	Aortic orifice.	Narrowing—Obstruction.
Systolic.	At ensiform cartilage.	Feebly transmitted.	Tricuspid orifice.	Incompetency—Regurgitation.
Systolic.	At left second intercostal space close to sternum.	Feebly transmitted.	Pulmonary orifice.	Narrowing—Obstruction.
Diastolic.	At junction of right second costal cartilage with sternum.	To midsternum—in course of sternum.	Aortic orifice.	Incompetency—Regurgitation.
Diastolic.	At left second intercostal space close to sternum.	In course of sternum.	Pulmonary orifice.	Incompetency—Regurgitation.
(Diastolic) presystolic.	Over body of heart.	To apex of heart.	Mitral orifice.	Narrowing—Obstruction.
(Diastolic) presystolic.	At ensiform cartilage.	Feebly transmitted.	Tricuspid orifice.	Narrowing—Obstruction.

Acute Pericarditis.

What are the distinguishing features of acute pericarditis?

Acute pericarditis may develop in association with rheumatism, infectious diseases, nephritis, or pleurisy, endocarditis, or other adjacent disease; it may also result from exposure to cold or from blows upon the chest. It is attended with pain in the precordial region, and sometimes in the epigastrium; considerable elevation of temperature and other febrile symptoms; a sense of anxiety; dyspnea; irregularity and increased rapidity of the heart; irritable cough; possibly headache, vertigo, delirium, nausea, and vomiting. The gastric phenomena may obscure the actual condition, or the cerebral symptoms may suggest meningitis; careful inquiry and examination will, however, reveal the inflammation of the pericardium.

What are the physical signs of acute pericarditis?

In the *first stage*, the characteristic sign is a rubbing *friction-sound* heard close to the ear at one or more points in the precordia, synchronously with one or both sounds of the heart. The sound is to be distinguished from an endocardial murmur by its quality, its superficiality and by increase of distinctness caused by pressure with the stethoscope.

In the *second stage*—that of *effusion*, a murmur is no longer heard, except perhaps above the fluid at the origin of the great vessels. The cardiac *impulse* is feeble, extended and wavy. The area of *percussion-dulness* is increased, and presents a peculiar outline—being *triangular*, with the base below. At the apex and over the body of the heart, the *sounds* are heard but feebly or not at all, while at the base they may be heard with ordinary distinctness.

In the *third stage*, the fluid has disappeared, and murmurs may again be heard. In the course of time, however, the opposite surfaces of the inflamed membrane become adherent, so that the pericardial cavity is in whole, or in part, obliterated. Adhesions to the chest-wall give rise to localized retraction, and to *systolic dimpling*. The heart in turn becomes enlarged.

How are acute pericarditis and acute pleurisy to be differentiated?

Pericarditis and pleuritis arise under similar conditions. The two may be associated. The physical phenomena of both are analogous, differing, however, in degree, extent and situation. The friction-sound of pleurisy is heard synchronously with the respiratory movements, that of pericarditis synchronously with the cardiac movements—the latter is thus the more frequent. If doubt arise, the breathing should be suspended for a short time: a pleural friction disappears. The percussion-dulness occasioned by an effusion into the pericardium is situated in the precordia, and has a characteristic triangular outline; the percussion-dulness of an effusion into the pleural cavity involves the lateral and posterior aspects, as well as the anterior aspect of the chest.

Acute Endocarditis.

What are the characteristics of acute endocarditis?

Acute endocarditis develops in the course of infectious diseases and of nephritis and chorea, but more especially in connection with acute rheumatism. It is characterized by a sense of distress in the precordia, palpitation and increased frequency of action of the heart, sometimes with irregularity, elevation of temperature, dyspnea, slight cough and an anxious expression. In addition, the face may be flushed; there may be chills, headache, vertigo, delirium, icterus, irritability of the stomach, and diarrhea. The characteristic sign of acute endocarditis is a soft, blowing, systolic murmur, though it is possible for endocarditis to exist without the detection of a murmur.

How is the murmur of an acute endocarditis to be distinguished from a murmur the result of a past endocarditis?

The murmur of acute endocarditis is soft, inconstant in its seat, unattended with enlargement of the heart and associated with the fever, precordial anxiety and other symptoms of acute endocarditis; a murmur dependent upon a past endocarditis is harsher, fixed in seat, unattended with fever, but associated with cardiac enlargement and a history of one of the conditions that may cause endocarditis or of a remote attack of endocarditis.

ACUTE ENDOCARDITIS.	CHRONIC VALVULAR DISEASE.
The murmur is soft and inconstant in seat and occurrence.	The murmur is harsher, fixed in seat and constant in occurrence.
Unattended with enlargement of the heart.	Attended with enlargement of the heart.
Associated with fever, precordial anxiety and other symptoms of acute endocarditis.	Not associated with fever or other symptoms of an acute condition.
No history of a remote acute endocarditis.	Remote history of acute endocarditis.
Associated with rheumatism or other cause of acute endocarditis.	Absence of an immediate cause of acute endocarditis.

How is acute endocarditis to be distinguished from acute pericarditis?

The pain of pericarditis is likely to be more severe than that of endocarditis. The murmur of pericarditis appears closer to the ear than that of endocarditis; the former is a friction-sound, diastolic as well as systolic; the latter is blowing in character and only systolic. When a pericardial effusion exists, it presents a peculiar area of dulness on percussion, the heart-sounds at the apex are enfeebled or they may be absent, and there is a diffused, feeble and wavy impulse, not found in endocarditis.

Mycotic Endocarditis.

What are the symptoms of mycotic or ulcerative endocarditis?

In addition to the ordinary symptoms of acute endocarditis, the *mycotic form* is marked by high temperature with decided oscillations, chills, sweats, the evidences of septic infection, ecchymoses, prostration, a typhoid condition and albuminuria. Mycotic endocarditis is usually engrafted upon preëxisting valvular disease. Septic vegetations may form and ulceration and perforation occur. Embolism is among the threatening dangers.

Myocarditis.

What are the symptoms of myocarditis?

Some degree of myocarditis necessarily attends pericarditis and endocarditis. *Inflammation of the muscular structure of the heart* may occur in the course of rheumatism or in association with septicemia or pyemia. The condition does not give rise to definite signs or symptoms apart from those of the inflammation of the pericardium or endocardium with which it is commonly associated.

What are the features of interstitial myocarditis?

Interstitial myocarditis may be a result of acute inflammation of the heart or form part of a general fibroid degeneration.

The action of a heart so affected is likely to be irregular and intermittent; one or both sounds may be duplicated, while the second is likely to be accentuated.

Hydropericardium.

What are the characteristics of hydropericardium?

When not the result of pericarditis, an *effusion into the pericardium* is usually part of a general dropsy. It presents *physical phenomena* indistinguishable from those of pericarditis in the stage of effusion, but lacks the febrile symptoms as well as the friction-murmurs of the first or of the third stage. Evidences of general dropsy, such as edema, pleural and peritoneal effusions, and symptoms or signs of the causative disease—nephritis, for example—will be found if looked for.

Heart-Clot.

What are the symptoms of heart-clot?

The blood may coagulate in the cavities of the heart in the course of endocarditis, of diseases in which the coagulability of the blood is increased, such as pneumonia, and when from any cause the action of the heart has become enfeebled. Under such circumstances the surface of the body becomes cold and livid, there is intense dyspnea, the action of the heart becomes rapid, feeble and irregular, a faint murmur may be heard over the organ and the area of cardiac percussion-dulness is increased. There is great anxiety, nausea, vomiting, nervous excitement, delirium, venous turgidity and attacks of syncope.

Thoracic Aneurism.

What are the symptoms of thoracic aneurism?

The symptoms and physical signs of *thoracic aneurism* vary with the location of the aneurism. Aneurisms of the aorta are the most common. The frequency of aneurisms of the aorta diminishes with the distance from the heart. The *characteristic manifestations* of an aneurism are the existence in the course of a bloodvessel of a *pulsating expansile tumor*, attended with a *thrill* and *bruit;* on percussion, a circumscribed *area of dulness* will be found to correspond with the area of expansile pulsation, over which a thrill can be perceived with the palpating hand, and a systolic bruit can be heard on auscultation. Other *symptoms* depend upon the compression of adjacent structures. There may thus be a peculiar metallic cough, with or without expectoration; dyspnea; dysphagia; irregularity of the action of the heart; venous stagnation and edema; manifestations of derangement of the cerebral circulation; localized sweating; dilatation or contraction of one pupil; paralysis of one or both vocal bands; enfeeblement of respiratory murmur over certain areas, from obstruction of a large bronchus; inequality of the radial pulses; neuralgic pain and erosion of bone.

How is an intra-thoracic tumor to be distinguished from an aortic aneurism?

An aneurism develops only in the course of a bloodvessl; a tumor of any other sort is not so restricted. The former presents pulsation, expansion, and usually thrill and bruit; the latter may rise and fall if seated over an artery, but does not expand or present a thrill or bruit. The sphygmographic tracing (taken *in loco*) of a solid tumor situated above a normal artery is that of the normal artery; the curve given by an aneurism is abnormal and often characteristic. Intra-thoracic tumors are usually either secondary or give rise to metastases. If the tumor were a gumma, the diagnosis would depend upon a history of syphilis or upon other manifestations of syphilis, together with a recession of the symptoms on appropriate treatment. Tuberculosis of the mediastinal glands would be but one manifestation of a general tuberculosis.

How is an abscess of the mediastinum to be distinguished from an aneurism of the aorta?

In addition to the features that distinguish all other thoracic tumors from aneurism, an abscess presents fluctuation if accessible, rigors, elevation of temperature, and sweating. There is a history of traumatism or of pyemia.

How is incompetency of the aortic valve to be distinguished from an aneurism of the aorta?

Incompetency of the aortic valve may be attended with marked pulsation in the course of the aorta, with a thrill and bruit, but the evidences of tumor and the consequences of compression are wanting; the radial pulses are of the Corrigan or water-hammer type, but are equal; while the second sound of the heart at the base is accompanied or replaced by a murmur transmitted downwards in the course of the sternum.

How is a dilated auricle to be distinguished from an aortic aneurism?

An enlarged auricle may give rise to pulsation at the base of the heart, but not to the compression-phenomena of an aneurism, not to the thrill or bruit, beyond the sounds generated in the heart itself as a result of the lesion that has led to the enlargement of the auricle: usually obstruction at the mitral orifice with a presystolic murmur.

Arterio-Capillary Fibrosis.

What is arterio-capillary fibrosis?

Arterio-capillary fibrosis is a degenerative process of the walls of the smaller bloodvessels, due to the long-continued circulation in the blood of irritants, such as alcohol, lead, the poisons of syphilis, of gout, of rheumatism, and of infectious diseases. Vaso-motor spasm and hyperplasia of the connective tissue of the arteries and of the intercellular elements of parenchymatous organs take place. The coats of the vessels become thickened and the size of organs becomes increased. From secondary contraction of the newly-formed fibrous tissue, the lumen of the arteries becomes narrowed and the organs become reduced in size. The circulation is curtailed and nutritive processes are interfered with; atrophy may result; thrombi may form *in situ.* Sclerotic vessels are prone to rupture, in the brain, in the retina, and elsewhere.

What are the symptoms of arterio-capillary fibrosis?

The diagnostic symptoms of arterio-capillary fibrosis or arteriosclerosis are those of vascular spasm, impeded circulation, and impaired nutrition: shortness of breath, vertigo, mental impairment, nervous derangement, irregularity of cardiac action, resistant radial arteries, prominent, tortuous temporals, increased arterial tension. Edema may be present. The *urine* becomes increased in quantity and may contain a trace of albumin and an occasional tube-cast. Numbness and coldness of the extremities are common. The *nails* are sometimes bluish from impeded circulation. The *knee-jerk* is often exaggerated. Transient local palsies sometimes occur. Angina pectoris and pseudo-angina are not uncommon. The action of the *heart* may be arhythmical, the sounds duplicated; the second or arterial sound is usually accentuated. An hypertrophied left ventricle, without valvular disease of the heart, should be suggestive of the existence of vascular disease. Chronic interstitial nephritis is a frequent result of arterio-capillary fibrosis, or of the same causes that give rise to the vascular changes. Cerebral and retinal thrombosis or hemorrhage may be *complications.*

Symmetrical Gangrene—Local Asphyxia.

What are the symptoms of symmetrical gangrene?

Raynaud's disease, *local asphyxia*, or *symmetrical gangrene* is an affection in which the fingers or toes on both sides of the body become cyanotic, edematous and numb; they may even undergo spontaneous gangrene. The pathology of the disease is not known; it is considered a vaso-motor neurosis, the persistent contraction of the peripheral capillaries giving rise to the symptoms.

The affection sometimes appears after exposure to severe cold. Its onset is occasionally marked by the occurrence of hematuria.

How are the symptoms of Raynaud's disease and those resulting from frost-bite to be differentiated?

The appearances presented by Raynaud's disease and those presented by frost-bite may be almost indistinguishable. Frost-bite is not likely, however, to exhibit the symmetry of distribution observed in cases of Raynaud's disease. The symptoms of frost-bite, moreover, usually recede when cold weather departs, while the manifestations of Raynaud's disease persist, though, perhaps, also aggravated by cold. Raynaud's disease is not necessarily dependent upon severe cold as a cause; while frost-bite is essentially so dependent.

THE RESPIRATORY SYSTEM.

How are affections of the upper air-passages (nose, pharynx, larynx and trachea) to be diagnosticated?

The diagnosis of affections of the upper air-passages is based, in part, upon the symptoms; but the only certain methods are those of *direct inspection*, or of *indirect inspection* by means of a suitable mirror. For both direct and indirect inspection, proper illumination is necessary. When direct daylight does not suffice, a reflector is employed, either with daylight or with artificial light. There are also apparatus for direct illumination by artificial light. Palpation with probe or finger is sometimes additionally necessary.

It is sometimes necessary to remove from the line of vision, by means of suitable retractors, certain structures, as the anterior palatine folds, the uvula, soft palate or epiglottis.

By *transillumination*, or transmission of light through the tissues, information is sometimes gained as to the comparative density of structure.

Direct inspection of the nasal passages by means of a speculum inserted by way of the nostrils is called *anterior rhinoscopy.*

Indirect inspection of the nasal passages by means of a mirror so placed in the pharynx, behind and below the soft palate, as to reflect an image of the posterior choanæ and neighboring structures, is called *posterior rhinoscopy.* The vault of the pharynx and the mouths of the Eustachian tubes are examined by the methods of posterior rhinoscopy.

Direct inspection of the pharynx is termed *direct pharyngoscopy.* To carry it out, only light to illuminate and a spatula to depress the tongue are required. Those parts of the pharynx inaccessible to direct inspection are examined by means of a mirror appropriately placed (*indirect pharyngoscopy*).

The laryngeal structures are rarely accessible to direct inspection. Sometimes a part of the epiglottis[1] may be seen, and in exceptional instances a glimpse of the supra-arytenoid structures has been obtained.

Laryngoscopy is practised by means of a mirror so placed in the pharynx, with the uvula lifted out of the way upon the back of the mirror, as to reflect an image of the deeper parts. In favorable cases, the interior of the trachea, to its bifurcation, may be seen. When the patient phonates, the superior face of the vocal bands and the structures above are reflected. When the patient inspires deeply, the internal face of the vocal bands and the structures below are additionally revealed. One endeavors to observe the contour, the color and the motility of the various parts, and to determine the presence or absence of secretion, ulceration, cicatrices, abnormal growths or foreign bodies.

To what conditions may difficulty of nasal respiration be due?

Difficulty of nasal respiration may be dependent upon enlargement, engorgement or distortion of the septum or of the turbinate bodies, upon the presence of foreign bodies, or of polypi or other neoplasms, or, especially in children, upon overgrowth of the glands of the vault of the pharynx (adenoid vegetations). The *diagnosis* can be made only by rhinoscopy, anterior and posterior, sometimes in conjunction with palpation.

---

[1] A very long epiglottis, visible on direct inspection, when the patient "gagged," has been mistaken by physicians for a red worm, and one hysterical patient, feeling the epiglottis with her finger, thought, because it was broad and hard, that it was a tapeworm.

What symptoms other than those directly referable to the nose may arise from the conditions that occasion difficulty in nasal respiration?

In addition to the local symptoms, the conditions that occasion difficulty of nasal respiration may be attended by anemia from deficient oxygenation, together with persistent cough, facial tic, headache, asthmatoid seizures, epileptiform convulsions, chorea, exopthalmic goiter, or various other reflex disturbances. The *diagnosis* is to be based upon a careful study of all of the phenomena and conditions, general and local.

What is a frequent, unsuspected cause of epistaxis?

An ulceration or erosion of the mucous membrane of the nasal septum, usually situated near the anterior inferior angle of the triangular cartilage, is a not uncommon cause of epistaxis. Malignant growth, especially sarcoma, which may involve the frontal sinuses and even be hidden from rhinoscopy is another condition worthy of mention. The bleeding may be insignificant in quantity, and might easily be considered of little moment.

Coryza—Acute Nasal Catarrh.

What is coryza?

Coryza or *acute nasal catarrh* is a superficial inflammation of the nasal mucous membrane, attended by sneezing and a profuse watery discharge from the nostrils. There is usually headache and sometimes more or less fever. The swollen membrane and more especially the engorged turbinate bodies impede respiration and modify articulation. In the course of a few days, the discharge becomes more viscid, often muco-purulent. Usually the attack subsides completely in a week. Conjunctivitis, pharyngitis, laryngitis, bronchitis and otitis media are among the *complications* and *sequelæ* sometimes manifested.

How is coryza to be differentiated from influenza?

Some authorities refuse to make a distinction between coryza and influenza. Coryza, with conjunctivitis or bronchitis, is sometimes epidemic. From fully-developed influenza, simple coryza, whether due to cold, to non-specific irritation or to infection, differs by the absence of the constitutional symptoms and of the profound depression characterizing the former disease.

Hay-Fever.

What is hay-fever?

Hay-fever, *hay-asthma*, *rag-weed fever*, *autumnal catarrh*, *June cold*, *rose-cold*, *idiosyncratic coryza*, *periodic vasomotor coryza*, are names applied to a group of symptoms developed in susceptible individuals as the effect of special irritants upon the mucous membranes of the eyes and air-passages, more especially of the nose. The *manifestations* vary much in severity and in constancy in different individuals, and in the same individual at different times, and comprise conjunctivitis, coryza, pharyngitis, laryngitis, bronchitis, asthma and gastric, enteric and renal crises. There is commonly intolerable itching of the eyelids and of the palate. Fever is not common; but when it occurs, it is irregular.

In some individuals the syndrome occurs when any irritating substance gains entrance to the nasal passages. In others the powder of certain drugs, such as ipecacuanha, or the pollen of certain plants, such as grasses, rag-weed, roses, is the *exciting cause*. The *predisposing cause* is usually a neurotic constitution, perhaps induced by excessive mental exertion or by undue indulgences; sometimes the presence of nasal abnormalities aggravates the symptoms.

In those in whom the attacks depend on special pollens, the manifestations necessarily recur *periodically*. In North America, the rag-weed is the most common provocative agent, and with most sufferers the attacks begin about the middle of August, and last until the cause has disappeared. June-colds are dependent on hay and roses.

How is hay-fever to be distinguished from simple coryza?

The distinguishing features of hay-fever are the invariability of its causation, and the severity and association of its symptoms; together with the rapid disappearance of symptoms on removal to a locality (usually the mountains or the seashore) where the provocative agency is not present.

Acute Laryngitis.

What are the symptoms of acute laryngitis?

Acute laryngitis usually results from exposure to cold, or from the inhalation of irritating fumes. It occurs deuteropathically in the course of many inflammatory and infectious diseases, whether general or of the respiratory or digestive organs. With great variations in the degree of severity, it manifests itself by laryngeal irritation, hoarseness or painful aphonia, painful deglutition, dyspnea and slight hawking cough, with mucous expectoration.

There may also be slight elevation of temperature.

Laryngoscopically, the laryngeal structures will be seen to be more or less reddened and swollen. If edema occur, the breathing will be labored and stridulous, and, as a result of the impediment to respiration, cyanosis may develop. Laryngoscopy or palpation will reveal the cause.

How is laryngitis to be distinguished from pharyngitis?

The only reliable method of discrimination is by inspection, both directly and by means of the mirror, as symptoms are very likely to be misleading, and the two affections not infrequently coexist.

In pharyngitis the alteration in voice—aphonia or hoarseness, of laryngitis is wanting. The difficulty and pain of swallowing are likely to be the less in laryngitis, and inspection will reveal the seat of the inflammation.

How does parotiditis differ from laryngitis?

In parotiditis the voice remains unaffected. In laryngitis there is no swelling about the face.

How does tonsillitis differ from laryngitis?

Inspection discloses the swelling of the tonsils and the absence of involvement of the larynx.

In tonsillitis the voice may be nasal, but it is not hoarse or lost; cough and expectoration are absent, while the difficulty of breathing may be great.

The difficulty and pain of swallowing are greater in tonsillitis than in laryngitis.

How does hysterical aphonia differ from acute laryngitis?

Hysterical aphonia, as a rule, sets in more or less suddenly; often in association with emotional disturbance, in a person, usually a female, presenting other manifestations of hysteria; and is unattended with pain, difficulty of breathing or elevation of temperature. Laryngoscopic examination will reveal the motor impairment of the vocal bands, and the non-existence of an inflammatory process. Recovery may take place as suddenly as did the onset. The attack is likely to be repeated.

Edema of the Larynx.

What are the symptoms of acute edema of the larynx?

Acute edema of the larynx may occur so insidiously as to produce death without giving rise to appreciable symptoms, or the symptoms may be sudden and overwhelming.

When edema of the larynx occurs in the course of other affections, or as a sequel, there may be the usual prodromes of inflammatory fever, but as a rule the onset is sudden, and the severity of the attack rapidly increases. There are local tenderness, dryness and heat in the throat, with a sensation as of the presence of a foreign body; more or less ineffectual cough; muffling or extinction of voice; difficulty of swallowing; difficulty of inspiration, with harsh stridor, occurring in paroxysms that increase in frequency and severity, and unless relief be afforded, result in expiratory difficulty and apnea.

Respiration is hurried and spasmodic; the *pulse* is small, frequent and irregular; the *temperature* is elevated; the *eyes* are prominent; the *face* is flushed and anxious, finally cyanotic.

When dependent upon acute disease, the suffocative attacks will be abrupt and violent, and will recur at intervals of a few hours.

When dependent upon chronic disease, the paroxysms may pass off, to recur irregularly, at progressively shorter intervals. Fever is absent, unless the underlying disease itself be febrile.

Simple inspection of the pharynx may reveal the swollen epiglottis projecting up behind the base of the tongue. *Palpation* with the finger may detect a soft, elastic, bladder-like swelling of the epiglottis, or of the ary-epiglottic folds; but unless cautiously practised it may induce a paroxysm of suffocation from the additional obstacle or irritation. *Laryngoscopic examination* at once reveals the cause of the symptoms.

What are the symptoms of chronic edema of the larynx?

Chronic edema of the larynx may be due to laryngeal disease, or to the usual causes of effusions. It gives rise eventually to progressive difficulty of breathing, paroxysmally aggravated, with perhaps some impairment of voice, and pain or difficulty in swallowing. The diagnosis is only to be made by laryngoscopy, in default of which palpation may perhaps be of service.

How is chronic edema of the larynx to be distinguished from asthma?

Principally by laryngoscopic examination. The paroxysms may exactly simulate asthmatic attacks. Suffocative paroxysms, due to laryngeal neoplasm, may likewise be mistaken for asthma if laryngoscopic examination be not made. Before the days of laryngoscopy many cases of sudden death were found to be due to laryngeal neoplasm.

Acute Tuberculous Laryngitis.

What are the characteristics of acute tuberculous laryngitis?

Acute tuberculous laryngitis is characterized by the *constitutional* symptoms of acute miliary tuberculosis, and *locally* by great pain in swallowing, by cough, dysphonia or hoarseness, and sometimes pain in respiration. *Laryngoscopic inspection* reveals peculiar thickenings of the epiglottis and other laryngeal structures, which are quickly followed by ulceration ; or there may be ulceration from the first. Tubercle-bacilli are sometimes to be detected in the secretions or in the débris of ulcers.

Laryngeal manifestations are sometimes observed before evidences of pulmonary tuberculosis can be detected ; but the latter, sooner or later, become manifest. A somewhat rapid fatal termination is the rule, but recognized instances of recovery are multiplying.

Laryngismus Stridulus.

What is laryngismus stridulus?

Laryngismus stridulus is a spasmodic affection characterized by contraction of the constrictor muscles of the larynx, giving rise to dyspnea, inspiratory stridor and a ringing, croupy cough. It occurs most frequently in rachitic children. The attacks are paroxysmal and usually nocturnal. The child may be awakened from sleep with a sense of suffocation and an appearance of lividity. The paroxysm lasts for a few minutes and terminates with a deep inspiration, attended with a crowing sound. Occasionally death results in the paroxysm ; perhaps, from incarceration of the epiglottis.

How does laryngismus stridulus differ from asthma?

Laryngismus stridulus is a disease of childhood ; asthma is rare in children.

Rickets predisposes to laryngismus stridulus ; asthma may be apparently idiopathic, or some recognized irritation, direct or reflex, may be discoverable. The wheezing and râles of asthma differ from the cough and stridor of laryngismus.

The asthmatic paroxysm terminates in abundant expectoration ; laryngismus stridulus subsides without critical phenomenon.

Laryngeal Vertigo.

What is laryngeal vertigo?

The name *laryngeal vertigo* is applied to a group of symptoms of rare occurrence that may appear in connection with recognized nervous diseases, as posterior spinal sclerosis or epilepsy, or without obvious explanation. The symptoms are not always the same, but the essential elements in association are pain or spasm of the larynx, vertigo and perhaps syncope. The occurrence of laryngeal vertigo should lead to careful search for symptoms or signs of the causative disorder.

Catarrhal Croup—Spasmodic Croup.

What is catarrhal or spasmodic croup?

Catarrhal or *spasmodic croup* is essentially a catarrhal laryngitis, associated with a tendency to spasm of the constrictor muscles of the larynx, causing paroxysms of suffocation. It is almost exclusively a disease of children. It may *follow* exposure, may be simultaneous with the prevalence of epidemics of influenza, of measles or of scarlatina. It may *begin* insidiously with slight hoarseness, or suddenly, with chill. It usually attracts attention at first toward evening; the voice will be hoarse, with a slight cough. In the middle of the night, the child will suddenly awaken, with crying and paroxysms of suffocation, and with a peculiar, ringing, metallic cough, which is termed croupy. The spasm may quickly pass away, or it may be repeated several times during the night and for three or four successive nights, the symptoms of ordinary laryngeal or laryngo-bronchial catarrh being manifested during the day. *Recovery* may take place, or high fever may set in with a firm, bounding pulse, flushing of the cheek, abnormal brilliance of the eye, and development of the obstructive symptoms of pseudo-membranous laryngitis.

How does catarrhal croup differ from laryngismus stridulus?

Laryngismus stridulus is a disorder of repeated occurrence, and comparatively chronic duration, occurring in rachitic children, and not associated with fever, cough or other symptom of inflammation. Spasmodic croup begins acutely, is of short duration, attacks healthy as well as rachitic children, and is associated with slight fever, cough and symptoms of catarrhal inflammation.

How are croup and edema of the larynx to be distinguished?

Apart from the revelations of laryngoscopy, croup is a disease of childhood principally—edema usually occurs in adults. In febrile cases the history of exposure to irritating fumes, or of other exciting cause of acute edema, the pain in swallowing, and the local tenderness; in other cases the absence of fever, and the knowledge of the existence of causative conditions; together with the absence of the croupal cough, and the purely inspiratory character of the stridor, are additional discriminative features of edema.

Membranous Croup.

What are the symptoms of membranous croup?

Membranous croup or *pseudo-membranous laryngitis*, whether an idiopathic disease or due to the extension or original formation of diphtheritic membrane in the larynx and trachea, is manifested by symptoms of mechanical and spasmodic or paralytic obstruction of the larynx and trachea, associated with more or less fever and the constitutional symptoms of diphtheria, if that disease be present. In some instances, the palate, tonsils and pharynx are involved in simple, exudative or diphtheritic inflammation. In severe cases, the process extends into the bronchi, and in some cases there is pneumonitis. There is a peculiar ringing cough, which gradually becomes muffled, with progressive muffling or extinction of the voice; and great difficulty in breathing, intensified in paroxysms, which may progress to suffocation. Evidences of the severity of the obstruction are the recession upon inspiration of the soft tissues above and below the sternum and the use by the child of the auxiliary muscles of respiration, in its efforts to obtain breath. There is extreme restlessness and agitation, with paroxysmal exacerbations, attended by protrusion of the eyeballs, distention of the nostrils, flushing of the cheek, grasping at supports.

Sometimes a child will sit up straight in bed and clutch at its neck, as if trying to pull away some obstruction.

Suffocative paroxysms, being partly spasmodic, are sometimes relaxed by the effects of the resulting carbonic acid poisoning, and with the production of cyanosis there comes a deep sighing expiration, followed by a deep inspiration, and a period of comparative quiet.

Flakes of membrane or even membranous casts of the trachea and bronchi may be expectorated. The *duration* may be short, from twenty-four to forty-eight hours; it may be prolonged to two or three weeks; usually, however, asphyxia or recovery occurs in from five to eight days. Convulsions often precede death.

How are membranous croup and retropharyngeal abscess to be distinguished?

Retropharyngeal abscess may be detected by inspection and palpation. The evidence of tuberculosis or syphilis, the history of previous infectious disease, may be suggestive of abscess. The voice is toneless but distinct in croup; in abscess it is nasal and indistinct. In abscess there are pain and difficulty in swallowing; these symptoms are absent from croup. In abscess there is stiffness of the neck, with tumefaction and pain on pressure externally; this is not the case in croup. In croup there is a peculiar cough and sometimes expectoration of false membrane; these are not present in abscess.

How do membranous croup and catarrhal croup differ?

In catarrhal croup fever and constitutional symptoms are slight. In membranous croup, fever is severe; and in diphtheria, toxemic symptoms are evident.

In catarrhal croup the obstruction to breathing is purely spasmodic, and disappears with relaxation of the spasm.

In membranous croup there is mechanical obstruction, continuing when the spasm has passed.

In catarrhal croup the voice remains clear, or at most becomes hoarse; the cough preserves its ringing, "croupal" character. In membranous croup voice and cough become toneless.

In membranous croup shreds of membrane or casts may be expectorated, and diphtheritic membrane is sometimes visible in the pharynx. Laryngoscopic inspection, when possible, will settle the diagnosis.

How is obstruction of the larynx, trachea or a bronchus by a foreign body to be distinguished from croup?

In the absence of the history, which would at once prevent error, the lack of fever and its concomitants, the absence of the peculiar cough, the variation in the symptoms, and finally laryngoscopic examination or the evidences of bronchial obstruction discovered on auscultation and percussion, permit the diagnosis of foreign body to be made.

Whooping-Cough—Pertussis.

What are the symptoms of whooping-cough?

Pertussis, or *whooping-cough*, is a contagious disease of childhood, attended with catarrhal symptoms, to which is added a peculiar cough, occurring in paroxysms and attended with a characteristic whoop. For several days there is coryza, accompanied with an acrid discharge, conjunctivitis, laryngitis and bronchitis. The cough then becomes paroxysmally explosive, a series of expiratory efforts being followed by a peculiar, ringing, inspiratory whoop. During these attacks the child may become livid and appear as if about to suffocate; sometimes vomiting is induced. The violence of the cough may cause ulceration of the under surface of the tongue on either side of the frenum; or there may be hemoptysis or epistaxis. The paroxysms are repeated with variable frequency. The disease may last many weeks, the severity and number of attacks progressively diminishing. It may be *complicated* by catarrhal pneumonia, which sometimes proves fatal. Bronchitis may long persist as a *sequel.*

What are points of discrimination between membranous croup and whooping-cough?

In whooping-cough there is little or no fever, no continuous dyspnea, no alteration of voice, and the child is usually up and about, unless there is pulmonary complication. In croup there is not the characteristic whoop.

What are the differential features of laryngismus stridulus and whooping-cough?

Laryngismus stridulus is a paroxysmal neurosis, most common in rickety children; whooping-cough is a contagious disease that attacks all children alike.

The cough of laryngismus stridulus is croupy; that of whooping-cough occurs as a series of explosive expiratory sounds, followed by a distinctive inspiratory whoop. During the intervals of freedom, whooping-cough presents the symptoms of bronchitis; laryngismus stridulus does not.

Chronic Laryngitis.

What are the symptoms of chronic laryngitis ?

Chronic laryngitis may be a result of repeated attacks of acute laryngitis; it may depend on nasal disease, gastric catarrh, dilated heart, constipation, or kidney disease, or other near or remote affection. It is common in those who use their voices improperly or immoderately, and in those who smoke to excess. It is characterized by hoarseness, persistent, irritable cough and discomfort in phonation and deglutition. Laryngoscopically the structures within the larynx are seen to be thickened, congested, covered by tough secretion; the vocal bands have lost their pearly, glistening appearance, and their motility may be impaired. In many cases chronic laryngitis is of tuberculous or of syphilitic origin.

How is chronic laryngitis to be distinguished from laryngeal neoplasms?

The only safe method of diagnosis is by means of laryngoscopy, and its neglect is criminal.

Chronic Tuberculosis of the Larynx.

What are the characteristics of chronic tuberculosis of the larynx?

In case of *chronic tuberculosis of the larynx* the subjective and objective symptoms may be simply those of ordinary chronic laryngitis, or there may be characteristic tumefactions, ulcerations or vegetations in the larynx, or all combined; with more or less pain and difficulty in swallowing and in respiration, and alteration of the voice. The signs of pulmonary tuberculosis are usually well advanced. Cases of recovery from the local disease in the larynx, or of its prolonged abeyance, are not rare.

How are paralyses, ulcerations, abscess, stricture and other conditions affecting the larynx and trachea, and giving rise to alterations in voice and difficulty in respiration, to be diagnosticated?

The only reliable method is laryngoscopy.

PHYSICAL DIAGNOSIS.

What are the methods of physical exploration?

In a *physical exploration*, systematically conducted, five means of investigation are employed: *inspection*, *mensuration*, *palpation*, *percussion* and *auscultation*.

What does physical exploration teach?

By physical exploration information is gained of physical or mechanical conditions, so far as these may influence the size, contour, movement, temperature, density, elasticity and acoustic relations of the structures examined. The nature of pathologic conditions cannot be directly determined from physical signs, but is to be inferred from the latter in association with all other phenomena in a given case.

What is to be learned by inspection?

By *inspection* the general appearance of an individual is noted—the height, the apparent relative nutrition, the color, the configuration, the movement, the state of the pupils, the expression.

Examining the chest more especially, its symmetry or asymmetry, its fulness or retraction; the frequency and character of the respiratory movements; the extent of the respiratory excursion; the situation, extent and vigor of the cardiac impulse are observed, and abnormal manifestations are looked for.

The chest is *distended* symmetrically in emphysema and usually in case of hydrothorax; pleural effusions and pneumothorax commonly occasion unilateral bulging.

The chest is *retracted* when the parietal and visceral pleuræ are adherent, and when the lung is shrunken, as in interstitial pneumonitis. The retraction is well marked after some forms of operative treatment of empyema, and when the lung is collapsed from any cause.

A healthy adult breathes from eighteen to twenty times in the minute. The normal respiratory excursion is curtailed and the frequency of respiratory action is accelerated by almost all diseases of the lung and pleura and also in case of peritonitis. The respiratory frequency is also accelerated when the pulse is quickened, as in febrile conditions. In case of pleurisy, the patient lies on the affected side, so as to reduce to a minimum functional movement of that side. Rapidity of breathing may be hysterical. Women breathe mostly with the upper portion of the chest; men with the lower.

What is to be learned by mensuration?

Mensuration determines with precision what inspection does approximately: the size, configuration, symmetry or asymmetry and respiratory excursion of the chest.

What is to be learned by palpation?

By *touch* or *palpation* one distinguishes elasticity from rigidity and resistance, investigates the existence of pain or tenderness, of edema, of moisture or dryness, of heat or cold. When the palm of the hand is applied to the chest of a speaking person a diffuse, vibratory sensation is perceived, usually slightly greater in intensity on the right side than on the left. This is known as the *vocal* or *tactile fremitus*.

It is increased in conditions of condensation of the lung or thickening and adhesion of the surfaces of the pleura. It is diminished or lost over collections of fluid in the pleura. Variations in vocal fremitus correspond pretty closely with variations in vocal resonance. Sometimes a pleuritic friction-rub can be felt.

How is percussion performed?

Percussion may be *immediate* or *mediate*. In the former a sharp blow is struck with the tips of the fingers bunched together, or with the palmar surface of the extended fingers.

Mediate percussion is performed by means of a thin, flat plate of ivory or of hard rubber (called a pleximeter), applied over the part to be examined, and a small rubber-tipped hammer (called a plexor).

For many purposes it is preferable to use the extended finger of one hand as a pleximeter, and the index or middle finger (or both) of the other hand as a plexor. Percussion is said to be *weak* or *strong*, *superficial* or *deep*, according to the energy of the blow of the plexor.

What is to be learned by percussion?

Percussion gives information as to the relative distribution of gases (usually air), fluids and solids in the structures examined. Attention is paid to the *quality* (or *timbre*), the *pitch* and the *intensity* of sounds.

Percussion of the healthy chest elicits a sound called *clear*, representing the *normal*, *pulmonary*, *vesicular resonance*. This may be *impaired* by increased density of the pulmonary tissues or of superjacent structures—as in interstitial pneumonitis, in pleural thickening or when the chest-wall is thickened.

Percussion-dulness is dependent upon a high degree of condensation—as in the solidification of pneumonia, or at the late stage of tubercle-formation.

The sound elicited over solid viscera, as the liver, heart or spleen, or over serous effusions—as in hydrothorax or hydropericardium, or in ascites, is *flat*.

The sound occasioned by air (or gas) in inclosed spaces larger than the alveoli of the normal lung may be *hyper-resonant* or *tympanitic*—as in vesicular emphysema or over the intestines.

An *amphoric* or *metallic* sound is elicited by percussion over large closed cavities, with tense walls, containing air—as a distended stomach or a large vomica in the lungs.

A *cracked-pot* or *cracked-metal* sound is occasioned by percussing over a cavity of some size, with a small opening, through which air escapes—as in the case of a pulmonary cavity communicating with a bronchial tube.

The percussion-sounds may display alterations in *degree* or *pitch*, and in *intensity* or *volume*.

Increased density gives heightened pitch—hence a dull sound is higher in pitch than a clear one. The pitch of tympanitic sounds varies. As a rule, it is higher than that of the normal pulmonary resonance. Other things being equal, the greater the volume of matter set in vibration the greater the intensity of the sound.

By mediate percussion with the fingers a sense of *elasticity*, or of *resistance*, can be appreciated.

Sometimes auscultation is practised by one while a second at the same time practises percussion—so-called *auscultatory percussion*.

The percussion sounds elicited during full-held inspiration differ from those elicited during full expiration—so-called *respiratory percussion*.

How is auscultation practised?

Auscultation, like percussion, may be *immediate* or *mediate*. In the former, the ear is directly applied to the part to be auscultated; in the latter, auscultation is performed through the mediation of a stethoscope. Stethoscopes are monaural or binaural. Each has its advantages. The student should become familiar with all of the methods of auscultation.

What is to be learned by auscultation?

Auscultation gives information as to the movement of air and fluids, as to the comparative calibers and lengths of the tubes through which the air passes, and as to the presence in the path of the air-current of matters capable of acting the part of reeds in the production of musical tones.

Listening to the normal respiratory sounds one hears a soft, breezy inspiration—the *normal vesicular murmur*, followed by a scarcely audible, briefer expiration.

These sounds may be *exaggerated*—as they normally are in children; hence they are then called *puerile*.

The respiratory sounds are also *intensified* in a lung, or in a portion of lung, performing an excess of function—as after violent exercise, or, compensatorily, when the function of the other lung, or of a portion of the same lung, is interfered with by condensation or compression.

The respiratory murmur is *enfeebled* or wanting when there is an obstruction to the circulation of air in the lung—as in incipient pulmonary tuberculosis, and in occlusion of the air-passages—as by intrathoracic aneurism, or when the air vesicles have lost their elasticity—as in emphysema.

The respiratory murmur is rendered *harsh* and is *heightened in pitch* when the bronchial tubes are thickened, and their caliber is narrowed—as in bronchitis. Under similar conditions, *rhonchi* or *dry râles* may be heard—*sibilant*, if generated in the small tubes; *sonorous*, if generated in the large tubes. Sometimes the sounds are *high-pitched* and *wheezing*.

If the vesicular murmur is but partially obliterated, the breathing is called *vesiculo-bronchial*; or if the bronchial element predominates, *broncho-vesicular*. When occlusion of the alveoli entirely obliterates the vesicular quality, the breathing is said to be *bronchial* or *tubular* or *blowing;* these terms respectively indicating progressive encroachment upon the integrity of the smaller air-tubes. In case of a cavity in the lung, the breathing may be *cavernous;* if the cavity have a tense wall and a small orifice of communication with a bronchus, the sound transmitted to the ear is *amphoric*.

The presence of secretion in the bronchial tubes occasions *râles*, usually qualified as *moist: subcrepitant*, if in the smaller tubes, *mucous* if in the larger.

The presence of fluid in a vomica may give rise to *bubbling* or *gurgling*.

The separation of the adherent surfaces of pulmonary alveoli lined by viscid secretion, as in pneumonia, gives rise to the *crepitant râle*. When isolated softening of pulmonary tubercles takes place, a sound is generated comparable to that produced when salt is thrown upon fire, or when a few hairs are rubbed together between the fingers—so-called *crackling.*

When moist râles are heard through solidified pulmonary tissues they are transmitted with a peculiar clearness and with a sort of ringing character. They are then called *consonating.*

In the first stage of pleurisy, before effusion has taken place, and in the third stage, after the fluid poured out has been absorbed, a *coarse crackling*, or *creaking*, or *rasping* sound of pleural *friction* may be heard.

In case of pneumothorax resulting from the perforation of a pulmonary vomica into the pleural cavity, vibration of the fluid that has been effused sometimes gives rise to *metallic tinkling.* Under similar conditions, or in case of a large vomica containing considerable fluid, vigorous shaking of the patient, with the ear applied to the chest, may elicit the sound of splashing—the so-called Hippocratic *succussion sound.*

When the ear is applied to the normal chest of a speaking individual a confused sound is heard—the *vocal resonance.*

If fluid is effused into the pleural cavity the transmission of the voice is intercepted below the upper level of the fluid. If the pulmonary tissues are solidified, as in pneumonia and tuberculosis, the resonance is increased, constituting *bronchophony.* When spoken language and whispers can be distinguished, the phenomena are termed *pectoriloquy* and *whispering pectoriloquy* respectively. The latter, if circumscribed, is usually indicative of the existence of a cavity in the lung.

When, in the course of pleurisy, a small quantity of fluid has been poured out the voice is transmitted above the level of the fluid with a peculiar bleating character, constituting *egophony.*

Not only may the character of the breath-sounds be altered, but their *rhythm* may deviate from the normal. Thus, expiration may be *prolonged*, as when, as in emphysema, the elasticity of the walls of the alveoli is diminished; or the expiratory sound may be *prolonged* or *jerking*, as when, as in the early stages of pulmonary tuberculosis, there is some obstruction to the exit of air.

Acute Pleurisy.

What are the symptoms of acute pleurisy?

Acute pleurisy may set in with a chill and sharp pain in the side, aggravated by the respiratory movements. The dyspnea may be slight or considerable. The *temperature* rises moderately high; the *breathing* is feeble, shallow and rapid. There is slight irritative cough, and scanty, frothy expectoration. In the course of a few days the symptoms subside, and at the end of a week or ten days the patient is well. Acute pleurisy may occur as a primary condition. It may follow traumatism or exposure to cold. Quite commonly it is secondary to inflammation of adjacent structures, especially of the lungs. It also occurs in the course of various infectious diseases of nephritis and of rheumatism. Pleurisy may be "dry" or attended with effusion. Effusion gives rise to respiratory and circulatory embarrassment proportionate to the volume of fluid poured out.

The fluid poured out may not be readily absorbed. On the contrary, it may remain obstinately persistent. In the course of time, it may become purulent; under other circumstances, it is purulent from the outset; in either case the condition is known as *empyema*. More commonly, the fluid is absorbed and the two layers of the pleura become adherent, with obliteration of the pleural cavity; in the progress of the case considerable thickening takes place, followed in turn by retraction of the chest. When the effusion is purulent there are repeated rigors, fever, sweats and emaciation; otherwise the health may be preserved, unless the chronic pleurisy is tuberculous. By the action of the heart, pulsation may be imparted to a collection of fluid in the left pleural cavity, so that an aneurism may be simulated. When the collection is purulent, the condition is designated *pulsatory empyema*.

What are the physical signs of acute pleurisy?

For convenience of study, the physical signs of acute pleurisy may be considered in *three stages*. In the *first*, or *plastic stage*, as the surfaces of the two layers of the pleura, roughened by exudation, slide over one another in inspiration and expiration, a harsh, creaking sound is heard, the vibration occasioning which may sometimes also be appreciable on palpation—the so-called *friction-sound* or *rub*. The *breathing* is shallow, feeble and rapid ; the movements of the affected side being restrained in the greater degree in order to mitigate the pain.

In the *second stage*, or *stage of effusion*, serum in variable quantity, possibly mixed with blood, is poured out into the pleural cavity. The corresponding lung is pushed upward and backward, and the heart, liver, spleen and diaphragm may be displaced. One side of the chest appears larger than the other, and the interspaces corresponding to the situation of the effusion are abnormally prominent. The *cardiac impulse* is visibly displaced. The *respiratory excursion* is limited. Below the upper level of the fluid the *percussion-note* is flat ; above, it is subtympanitic. Through the effusion the *breath-sounds* are heard feebly and indistinctly ; the *voice-vibrations* are not transmitted to the palpating hand ; nor, as a rule, to the auscultating ear, though exceptionally bronchophony may exist. To the ear applied to the chest above the level of the fluid the *voice* may be transmitted with a peculiar bleating quality—constituting egophony.

In the *third stage*, the effusion has been absorbed and the apposed layers of pleura again come in contact. The friction-sound of the first stage returns. Ultimately the pleura may be restored to its original condition or its apposed surfaces may become adherent and thickened, giving rise to retraction of the chest and percussion-dulness.

How does acute pleurisy differ from croupous pneumonia?

Pleurisy usually accompanies pneumonia. The signs of the former, however, should not be permitted to obscure the existence of the latter. Neither the local nor the general symptoms of pleurisy are so profound as those of pneumonia. While pneumonia may then present the symptoms of pleurisy, pleurisy does not present the blood-streaked expectoration, the crepitant râle, the blowing breathing, the deficiency of the chlorides in the urine, or the critical termination of pneumonia.

The percussion-note of pneumonia is dull ; that of pleurisy, when an effusion exists, is flat. The dulness of pneumonia is usually over a lower lobe; that of pleurisy is universally at the base. In pleurisy, the breath-sounds are heard feebly, or not at all, through the effusion; in pneumonia the breathing is bronchial. Vocal resonance and fremitus are increased in pneumonia; they are diminished or absent in pleural effusion; above the fluid, however, a bleating sound is transmitted to the auscultating ear. Simple, uncomplicated pneumonia is unattended with friction-sounds and occasions no displacement of adjacent viscera.

How are intercostal neuralgia and acute pleurisy to be differentiated?

The pain of intercostal neuralgia may simulate that of acute pleurisy, and give rise to rapid, shallow, feeble respiratory movements. Intercostal neuralgia is paroxysmal and unattended with friction-sound, dulness on percussion or fever; it occurs in anemic individuals with neurotic tendencies, and may be attended with a unilateral herpetic eruption in the course of the nerve affected. In addition there are a number of tender points.

How are a pleuritic effusion and an hydatid cyst of the liver to be differentiated?

When an hydatid cyst of the liver attains proportions sufficient to give rise to definite physical phenomena, these will appear in a region beyond that in which the signs of a pleuritic effusion on the right side are found. The history of an acute attack is wanting, while it is present in pleuritic effusion. Neither in case of pleuritic effusion, nor in case of hydatid of the liver, is the percussion-dulness confined to the right hypochondrium; in the one it extends rather upwards, in the other downwards; in the latter it is associated with fluctuation. In a case of hydatid cyst it may by percussion be possible to elicit the characteristic thrill or fremitus. In a case of pleuritic effusion the breath-sounds and the vibrations of the voice are feebly transmitted; breath-sounds, vocal resonance, and fremitus are unaltered in hydatid cyst of the liver. Egophony is characteristic of the presence of fluid in the pleura; it is thus not present in case of hydatid cyst of the liver. Exploratory puncture may determine the presence of hydatid hooklets, the detection of which places the diagnosis beyond doubt.

How are a pleuritic effusion and abscess of the liver to be differentiated?

Symptoms of respiratory interference are naturally less conspicuous in case of abscess of the liver than in case of pleuritic effusion. Hepatic abscess may occasion tumefaction in the right hypochondrium; pleural effusion renders the chest asymmetrical from unilateral bulging. Pleuritic effusion impairs the transmission of the breath-sounds and of the vibrations of the voice, which is unaltered by an hepatic abscess. Egophony may be elicited in case of pleuritic effusion, but not in case of hepatic abscess. Rigors commonly attend an abscess of the liver; they only take place in cases of pleuritic effusion when suppuration has occurred. In case of hepatic abscess, there is usually a history of gallstone, of ulceration of the bowel or of pyemia; in case of pleuritic effusion, there is a history of an acute attack of pleurisy. Hepatic abscess and perihepatitis may, however, give rise to pleural effusion by extension.

How are pleuritic effusion and abscess of the spleen to be differentiated?

Abscess of the spleen is most common as a manifestation of pyemia; pleuritic effusion is a sequel of an acute pleurisy. The symptoms of respiratory derangement, a necessary part of pleuritic effusion, are subordinate in case of splenic abscess. Repeated rigors occur in abscess of the spleen; rigors occur in case of empyema, but not when the effusion is not purulent. Egophony may be developed when fluid is present in the pleural cavity, but not in case of splenic abscess.

How is an intra-thoracic tumor to be distinguished from chronic pleurisy?

Tumors in the chest may arise from the pleura or from the lung; they may develop in the mediastinum; or they may be aneurismal. Malignant tumors are secondary to growths elsewhere—an element in diagnosis. They give rise to circumscribed areas of dulness on percussion, not necessarily limited to one side; to enfeebled breathing and perhaps friction-sounds;

in their physical signs, they more closely resemble encysted pleurisy than ordinary chronic pleurisy. When actually complicated by pleurisy, the diagnosis may not be possible. Aneurisms occur in the course of the aorta and large vessels, and, in addition to the evidences of tumor, are accompanied by thrill, bruit and pulsation.

How are a pleuritic effusion and hydrothorax to be differentiated?

Hydrothorax occurs as a result of cardiac insufficiency or as a part of a general dropsy, from nephritis for instance.

In contradistinction from pleuritic effusion, the effusion of hydrothorax is bilateral. In the former there has been an antecedent pleurisy; in the latter there are other evidences of cardiac failure or of a general dropsy.

How is a pericardial effusion to be distinguished from a pleural effusion?

A pericardial effusion occurs under circumstances similar to those that give rise to a pleural effusion—as a result of inflammation or as a part of a general dropsy. The position and outline of the percussion-dulness to which a pericardial effusion gives rise, however, are entirely different from what is found in a case of pleural effusion; nor are the breath-sounds notably interfered with, while the circulation is embarrassed and the heart-sounds are almost obliterated at the cardiac apex.

Acute Bronchitis.

What are the symptoms of acute bronchitis?

Acute bronchitis results from exposure to cold, the inhalation of irritating fumes, or as a secondary disorder in the course of fevers, rheumatism or heart-disease. There is irritative cough, and, at first, scanty mucous expectoration, which subsequently becomes more copious and muco-purulent; slight elevation of temperature; increased frequency of respiration; some dyspnea; retro-sternal pain, and mild constitutional symptoms.

What are the physical signs of acute bronchitis?

The *chest* is not deformed and expands well. The *percussion-resonance* is vesicular. The *breathing* is harsh at first; dry râles, sonorous and sibilant, are heard; subsequently large and small moist râles. *Vocal resonance* and *fremitus* are not perceptibly altered.

How does acute bronchitis differ from acute miliary tuberculosis?

In acute miliary tuberculosis the dyspnea is greater, the temperature is higher, with greater oscillations, the breathing is more rapid, and the symptoms are more profound than in acute bronchitis. The further progress of the case clears up any possible doubt. Recovery from acute bronchitis is the invariable rule. Pulmonary consolidation and softening, percussion-dulness and fine moist râles, emaciation, hectic fever, gradual failure of the vital powers and ultimately death mark the usual course of acute miliary tuberculosis.

Chronic Bronchitis.

What are the symptoms of chronic bronchitis?

Chronic bronchitis is usually a result of repeated attacks of acute bronchitis; it may manifest itself as a special susceptibility to acute bronchitis; at first it appears as a winter cough, subsequently becoming continuous. It may obstinately resist treatment; it is attended with a good deal of cough, copious muco-purulent expectoration, marked shortness of breath, and may in time give rise to emphysema, or to bronchiectasis. It is often attended with loss of flesh and strength.

What are the physical signs of chronic bronchitis?

If emphysema coexists the *chest* may be enlarged; otherwise it is not abnormal in size or form; the *respiratory excursion* is somewhat diminished; the *percussion-resonance* is little or not at all impaired; the *breathing* is harsh and may be feeble, the bronchial element preponderating. Coarse râles, moist and dry, are heard at all parts of the chest. *Vocal fremitus* and *resonance* are rather increased.

How is chronic bronchitis to be distinguished from interstitial pneumonitis?

A certain degree of bronchitis commonly attends interstitial pneumonitis; but the chest is likely to undergo retraction, which is not the case in bronchitis. If impairment of resonance attend chronic bronchitis, it is general and not well defined, while the dulness of interstitial pneumonitis is the more circumscribed and the more decided.

How does chronic bronchitis differ from pulmonary tuberculosis?

While chronic bronchitis may be attended with obstinate cough, muco-purulent, sometimes blood-streaked, expectoration, loss of flesh and strength, the physical signs are general in their distribution and not localized as in tuberculosis. The impairment of resonance is not so great in chronic bronchitis as it is in tuberculosis. The elevation of temperature observed in tuberculosis is wanting in bronchitis. Tubercle-bacilli are not found in the sputum of cases of simple chronic bronchitis.

Plastic Bronchitis—Fibrinous Bronchitis.

What are the characteristics of plastic or fibrinous bronchitis?

In addition to the phenomena of ordinary bronchitis, there are present in *plastic bronchitis* decided dyspnea and cyanosis, together with the expectoration of tough, fibrinous casts of the smaller bronchial tubes; there occur lancinating pains in the chest; and there may be bleeding from the nose and mouth.

Putrid Bronchitis.

What are the clinical features of putrid bronchitis?

In case of bronchitis with bronchial dilatation, accumulation of secretion may take place, with ulceration and inflammation of the bronchial mucous membrane, as a consequence of which expectoration is augmented, the breath and sputa possessing an offensive, fetid odor. To these phenomena fever and a typhoid condition may be added—and even pulmonary gangrene may supervene.

Bronchiectasis.

What is bronchiectasis?

Bronchiectasis consists in a cylindrical or saccular dilatation of the bronchial tubes, usually developed in the course of some condition attended with powerful or sustained expiratory efforts—such as chronic bronchitis. It may also result from contraction of the peribronchial tissues. In addition to the symptoms of the causative affection, the characteristic feature of bronchiectasis is the periodical occurrence of paroxysms of cough, attended with the expectoration of large quantities of muco-purulent secretion of offensive odor. The tubes filled with fluid may give rise in small areas of irregular distribution to *percussion-dulness* that disappears with the evacuation of the fluid. On *auscultation* large, coarse, moist râles and gurgling may be heard. If of moderately large size, bronchiectatic cavities may yield a hyper-resonant percussion-note, with blowing breathing and whispering pectoriloquy.

How does bronchiectasis differ from pulmonary gangrene?

The grave constitutional symptoms of gangrene are lacking in bronchiectasis. The expectoration of both is offensive, but that in gangrene is actually fetid. The sputum of gangrene contains shreds of pulmonary structure: that of bronchiectasis does not.

How does bronchiectasis differ from pulmonary abscess?

A sacculated dilatation of a bronchial tube containing fluid fulfils the physical conditions of an abscess communicating with a bronchial tube. The expectoration of a case of bronchial dilatation, however, has an offensive odor not present in abscess. The fever and sweats of abscess are wanting in bronchiectasis.

How are bronchiectasis and catarrhal pneumonia to be differentiated?

In the small areas of dulness of irregular distribution, bronchiectasis may simulate catarrhal pneumonia, but the former is wanting in the febrile phenomena and the constitutional depression of the latter disease. The association of bronchiectasis with chronic bronchitis is different from the occurrence of catarrhal pneumonia in children. The offensive sputa of the former are entirely wanting in the latter.

Capillary Bronchitis.

What are the symptoms of capillary bronchitis?

Capillary bronchitis, sometimes called *suffocative catarrh*, is an inflammation of the smallest or "capillary" bronchi, most common in children and in old persons, and usually secondary to bronchitis of the larger tubes. In addition to the symptoms of the latter affection, capillary bronchitis is attended with decided constitutional depression; the dyspnea is more profound, and cyanosis may be marked.

Cough is severe, while expectoration is scanty; the *pulse* is rapid, the *respirations* hurried, the *countenance* discolored, the expression anxious; fever is moderately high.

What are the physical signs of capillary bronchitis?

The *chest* is symmetrical. The *breathing* is rapid and shallow. The *percussion-resonance* is vesicular, though in spots it may be slightly impaired. When it exists, the impairment of resonance does not depend upon exudation, but upon atelectasis. The phenomenon may, therefore, be inconstant, and its location shifting. The *breath-sounds* are harsh, and in many places in both lungs small, mucous râles may be heard.

Vocal resonance and *fremitus* are slightly increased. The deviations from the normal are most marked at the bases of the lungs posteriorly.

How are capillary bronchitis and acute miliary tuberculosis to be differentiated?

Capillary bronchitis is a quite brief disease of the very young and the very old; acute miliary tuberculosis is a more prolonged disease of the young and of adults. Dyspnea, cyanosis and constitutional depression are early more decided in capillary bronchitis than in acute miliary tuberculosis. The fever of tuberculosis is more intense and vacillating than that of bronchitis. Localized areas of persistent percussion-dulness, followed by the moist râles of softening, may develop in the course of acute miliary tuberculosis, which is progressively fatal; recovery from capillary bronchitis is not uncommon.

Catarrhal Pneumonia—Broncho-pneumonia.

What are the symptoms of catarrhal pneumonia?

Catarrhal pneumonia, also called *lobular pneumonia*, or *broncho-pneumonitis*, may be apparently primary, but is often secondary to capillary bronchitis, as also to measles, influenza and other constitutional affections; it may result from the inspiration of infectious material, whether contained in the food or from septic surfaces in the respiratory tract.

It is attended with cough, muco-purulent expectoration, increased frequency of respiration, decided febrile phenomena that may be hectic, and constitutional depression. The course of the disease may be protracted.

What are the physical signs of catarrhal pneumonia?

The *chest* is symmetrical; *respiration* is shallow.

The *range of movement* is sometimes curtailed on one side.

Small, irregularly distributed patches of *percussion-dulness* may be found here and there over both sides of the chest, anteriorly and posteriorly; the *breathing* is harsh; fine and large moist râles may be heard; the *vocal resonance* and *fremitus* are intensified in patches corresponding, as a rule, to the areas of percussion-dulness.

How does catarrhal pneumonia differ from capillary bronchitis?

The gravity of the symptoms in both depends upon the extent of the disease. Other things being equal, capillary bronchitis is the more acutely dangerous affection, the depression sometimes being profound. The duration of catarrhal pneumonia is the longer. Its febrile phenomena are the more decided, and its temperature-range is marked by great oscillations. The impairment of percussion-resonance is more decided in catarrhal pneumonia than in capillary bronchitis; it is constant, and does not change its seat.

What are the means of distinguishing atelectasis from catarrhal pneumonia?

Atelectasis is the pre-natal condition of the lungs, before they have been inflated with air. It may also arise in the course of affections attended with inspiratory obstruction of the bronchial tubes, as in whooping-cough or capillary bronchitis, for instance, a plug of mucus acting as a ball-valve, allowing the expiration of air but preventing inspiration. It is said also to occur in parts of the lung functionally inactive. The condition is not an inflammatory one and is not necessarily attended with elevation of temperature, as is catarrhal pneumonia. The collapse is not persistent, but may successively involve different parts of the lung; so that the physical signs are not constant, but transitory as well as migratory.

How does catarrhal pneumonia differ from pulmonary infarction?

Catarrhal pneumonia does not set in with the acuteness and the severe pain of pulmonary infarction; nor is the expectoration as persistently blood-streaked in the former as in the latter. Catarrhal pneumonia is attended with decided febrile manifestations and is of considerable duration, while pulmonary infarction scarcely occasions fever and its symptoms soon subside. The detection of a source of an embolus would be strongly in favor of the existence of infarction.

Acute Croupous Pneumonia.

What are the symptoms of croupous pneumonia?

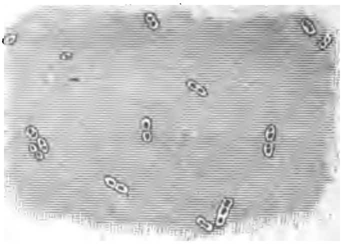
Croupous pneumonia, also called *lobar pneumonia*, and at one time *lung fever*, is an acute, infectious disease dependent upon the invasion of microörganisms (Fig. 22), often following exposure to cold, *setting in* suddenly with a chill, sometimes with convulsions, the *temperature* rising at once to 103° or 104° F., the *respiratory frequency* reaching 35, 40, 50 or more, while the *pulse* is not proportionately accelerated.

There is at first slight cough, with scanty expectoration of a viscid sputum, which soon becomes blood-streaked (rusty).

There is considerable dyspnea; the alæ nasi are retracted in breathing; and one or both cheeks are flushed.

The cough increases; the expectoration becomes more free, more hemorrhagic, less viscid, and presents the appearance of prune-juice. There is pain in the chest, often referred to the nipple of the affected side; usually involving the lateral or axillary region in addition. The pain is increased on attempting to make prolonged inspiration. The *lips* and *cheeks* may be cyanotic. The *tongue* is coated; the *appetite* is lost; *thirst* is increased; there may be jaundice, and, at the height of the attack, delirium.

FIG. 22.



Micrococci of croupous pneumonia. (Vierordt.)

Between the second and fifth days, herpes of the lips may appear. The symptoms continue for from five to seven or nine days, to subside by *crisis*. Profuse perspiration occurs; the temperature declines; and convalescence sets in.

The cough is still free; the expectoration copious and muco-purulent. During the attack the urine is deficient or wanting in chlorides, which return as resolution proceeds. Resolution may fail to take place, or it may be but partial; so that suppuration may ensue or chronic induration be a sequel. Death happens in a variable proportion of cases, not rarely from heart-clot or heart-failure.

Pleurisy is a common accompaniment of pneumonia. Occasionally large effusions are poured into the pleural cavity.

What are the physical signs of croupous pneumonia?

For convenience of description, pneumonia is divided into three stages: a stage of congestion, a stage of exudation, a stage of resolution.

What are the physical signs of the first stage of pneumonia, or the stage of congestion?

The aspect of the *chest* is unchanged.

The *breathing* is accelerated, becoming shallow and labored.

The *percussion-resonance* is little or not at all impaired.

The *respiratory murmur* is roughened over a circumscribed area, usually corresponding to a lower lobe. At the end of inspiration a fine, moist, crepitating râle may be heard.

Vocal resonance and *fremitus* are not perceptibly altered.

What are the physical signs of the second stage of pneumonia, or the stage of exudation?

The *respiratory excursion* is curtailed; the *breathing* is hurried.

The lower part of the *chest* on one side, corresponding to the lower lobe of the lung, may be a little fuller than elsewhere. Here the *percussion-note* is dull. Above, the note is hyper-resonant.

At the dull area, the *breathing* is bronchial in character. At other parts of the chest the breathing is puerile. The crepitant râle has disappeared.

Vocal resonance and *fremitus* are increased at the area of percussion-dulness. A pleuritic friction-sound may be heard and the signs of effusion may develop.

What are the physical signs of the third stage of pneumonia, or the stage of resolution?

The localized fulness of the *chest* may remain.

The *respiratory excursion* and the *expansion* have somewhat augmented.

Some degree of impairment of the *percussion-resonance* persists.

The *breathing* is yet bronchial, though not in the same degree as it was. The crepitant râle, which for a time had disappeared, can again be heard—the *crepitus redux;* other moist râles also now become discernible.

Vocal resonance and *fremitus* are still increased.

How is edema of the lungs to be distinguished from pneumonia?

Pulmonary edema may develop in the course of heart-disease, if the compensation be disturbed; in the course of nephritis or in the course of general asthenia; in subjects of these affections, it may follow exposure. It is not confined to one lung, but involves both alike throughout their entire extent.

Pulmonary edema may be uncomplicated, but is likely to be associated with effusions into the serous cavities and dropsy elsewhere. The sputum is frothy and abundant, but not rusty or like prune-juice. The cheeks are flushed in pneumonia; the face is pale or livid in pulmonary edema. Edema usually does not present febrile symptoms; the percussion-resonance scarcely suffers; large and small moist râles are heard all over the chest; the proportion of chlorides in the urine remains unaltered; while the brief duration of the condition, speedily terminating, as it does, in recovery or in death, and the knowlledge of the existence of a condition that may occasion it, make the diagnosis clear.

In what respects do catarrhal and croupous pneumonia differ?

Croupous pneumonia is sudden in onset; catarrhal pneumonia is rather commonly secondary to some other condition. Lobar pneumonia usually attacks robust adults; broncho-pneumonia, children, the aged, the debilitated. The sputum of catarrhal pneumonia is not blood-streaked, as is that of croupous pneumonia. Croupous pneumonia terminates in the course of seven or nine days by crisis; catarrhal pneumonia is a disease of long duration, with tardy convalescence. The physical signs of croupous pneumonia are well-defined, usually limited to one lung and to a lower lobe; the signs of catarrhal pneumonia are ill-defined, and irregularly disseminated.

How is passive or hypostatic congestion of the lungs to be distinguished from pneumonia?

Passive or hypostatic congestion of the lungs may develop in the course of acute or chronic debilitating diseases or as a result of heart-disease; it usually affects the bases and most dependent portions of the lungs. It is attended with increased frequency of breathing, impaired percussion-resonance, and the presence of fine râles. It differs from pneumonia in its association with the conditions that give rise to it; in the absence of fever, unless arising in the course of a febrile disorder; in its bilateral distribution; in the absence of blowing breathing; in its course and in its submission to appropriate management. If expectoration attend congestion of the lungs it is not hemorrhagic.

How is pulmonary infarction to be distinguished from pneumonia?

Coagulation of the circulating blood may take place in the right side of the heart as a result of increased coagulability or of retarded circulation, or a thrombus may form in a distant vein, and detached fragments of clot be swept into the lungs. The occurrence is attended with pain in the chest, dyspnea, and blood-streaked expectoration. Small areas of dulness on percussion can be detected, with enfeebled or bronchial breathing. There may be slight fever, from secondary inflammation. The condition differs from pneumonia in being secondary to a preexistent condition, in the circumscribed distribution of the physical phenomena on the part of the lungs, in the comparative absence of fever, and in the course of the disease. Pulmonary infarction is not of itself fatal. The danger is from the condition of which the infarction is but one result.

How is pulmonary infarction to be distinguished from pulmonary abscess?

Pulmonary embolism may result in either infarction or abscess. The outcome depends upon the presence or absence of pyogenic microörganisms. The immediate phenomena are the same in both instances. If suppuration take place, however, there are repeated chills, decided fever, with purulent expectoration. Pulmonary abscess is obviously the graver condition. The existence of disseminated suppuration is an aid in the diagnosis.

How is pneumonia to be discriminated from pulmonary tuberculosis?

Pneumonia is an acute disease, terminating by crisis in from five to nine days; the tendency of pulmonary tuberculosis is to be chronic; its duration is indefinite. The sputum of pneumonia may be profusely admixed with blood, but actual hemorrhage does not occur except in the peculiar form of pneumonia associated with influenza. The emaciation, the anemia, the sweats of pulmonary tuberculosis are not present in uncomplicated croupous pneumonia. The temperature of tuberculosis commonly displays wide variations; the temperature of pneumonia is of a continued type. Tuberculosis usually involves the upper portion of the lung, successively invading the remainder of the lung, and the opposite lung as well; pneumonia is usually unilateral and situated in a lower lobe. The physical signs differ in accordance with this peculiarity of distribution. It is not uncommon for pneumonia to be superadded to pulmonary tuberculosis.

Pulmonary Gangrene.

What are the clinical features of pulmonary gangrene?

Gangrene of the lung may result in the course of pulmonary tuberculosis, or of pneumonia; it may depend upon ulcerative communication of the lung with an adjacent septic process; it may be occasioned by the inspiration of septic matters from some portion of the respiratory tract; finally it may have its origin in a septic embolus. The symptom characteristic of pulmonary gangrene is the expectoration of a horribly offensive, stinking sputum, in association with a typhoid condition. Unless cavitation occurs, the physical signs are obscure; at best, they may be masked by those of the primary condition.

How is pulmonary gangrene to be distinguished from pulmonary abscess?

The physical signs and the local phenomena of pulmonary gangrene and of pulmonary abscess may be similar; but the expectoration in a case of abscess is copious and purulent, while that of a case of gangrene is brownish and horribly offensive and more likely to contain fragments of pulmonary structure. The signs of a cavity are more likely to attend abscess than to attend gangrene. Gangrene is more nearly certain than abscess to be fatal.

How are pulmonary tuberculosis and pulmonary gangrene to be differentiated?

Gangrene may complicate tuberculosis of the lung. It differs from uncomplicated tuberculosis by the characteristic odor of the breath and sputum and the absence of tubercle-bacilli from the matters expectorated.

Pulmonary Tuberculosis.

What are the varieties of pulmonary tuberculosis?

In the present state of knowledge pulmonary tuberculosis may, for practical purposes, be considered under two forms: *confluent* and *disseminated*. The lesion in both is essentially the same, the process in the one instance having a tendency to localization and chronicity, with slow extension; in the other to diffusion and acuteness. The first is commonly known as pulmonary phthisis, the second as acute miliary tuberculosis. Acute miliary tuberculosis most frequently involves several organs from the first, and is comparable, in many respects, to an acute infectious fever, such as one of the exanthemata. Localized tuberculosis may undergo retrogression and cicatrization by fibroid substitution, sometimes associated with calcareous deposits. On the other hand, it may terminate by softening (cheesy necrosis or caseation) leading to excavation, or the formation of vomicæ. Calcification, however, may occur, even after caseation has taken place.

Galloping consumption or *florid phthisis* is either an extremely rapid form of confluent tuberculosis, or a rapidly caseating subacute or chronic broncho-pneumonitis, on which tuberculosis has supervened.

Fibroid phthisis is a slow, localized tuberculosis in which cicatrization keeps almost even pace with tuberculous ulceration.

The fibroid conditions of the lungs found in miners, in steel-grinders, and in others exposed to the inhalation of fine particles of dust, will be considered as forms of interstitial pneumonitis, comparable to interstitial hepatitis and interstitial nephritis. Tuberculosis may supervene, running then the ordinary course.

What are the symptoms of pulmonary tuberculosis?

The course of ordinary chronic pulmonary tuberculosis may pathologically be divided into *three stages:* a *first* or *incipient stage*, in which the formation of tubercles begins; isolated, miliary nodules are localized at some part of the lung, most commonly at an apex; a *second stage*, of *consolidation*, in which the formation of tubercles has increased in number and density, but not correspondingly in extent, the tendency to localization continuing; and a *third stage*, of *softening*, in which, as a result of caseation and breaking down of the older tuberculous formations, cavities develop; while at the same time invasion of new areas takes place.

The *physical signs* in the different stages differ in accordance with the lesions. The *symptoms* of one stage, however, pass by imperceptible gradations into those of another.

The *onset* of the disease is usually insidious. The individual complains of a sense of lassitude and of an unusual readiness of fatigue. Exertion induces shortness of breath. There is a gradual failure of nutrition. The *color* fades; the *digestion* is deranged. Perhaps now a slight, irritating cough, attended with little or no expectoration, sets in. Sometimes, however, the first symptoms are observed after a neglected "cold," or supervene upon an attack of catarrhal pneumonia (so-called caseous phthisis). An occasional sense of chilliness in the back may be perceived. The *body-weight* diminishes. Perhaps at this time, unprovoked, the patient feels a tickling in the throat, perceives a salty taste, and before he realizes what is about to transpire he ejects a mouthful of bright-red, frothy blood. The *hemorrhage* may be repeated; at this stage it is not likely to be fatal. The cough becomes more aggravated; it occurs in paroxysms, the severity of which may induce vomiting. The *sputum* is abundant and muco-purulent; it contains elastic fibers and tubercle-bacilli. The dyspnea has become decided. Emaciation progresses; evening fever appears; debilitating night-sweats occur. Hemorrhages become more common and more profuse. The *cheek* is flushed, the *eye* bright, the *intelligence* quickened, the *mind* hopeful. The *hair* becomes straight and prematurely gray. *Digestion* fails. Diarrhea becomes troublesome. Atrocious pains in various parts of the chest indicate the existence of accompanying pleuritic processes. The ends of the fingers become enlarged and bulbous, the nails incurvated. The anemia is profound; the *lower extremities* become edematous. The patient must take to his bed. In the last stages of the disease, the patient may suddenly be seized with agonizing pain in the side, increased dyspnea, and a sense of impending death. One side of the chest becomes enlarged; the percussion-resonance is tympanitic; the breathing is but feebly transmitted; and metallic tinkling may be developed. *Pneumothorax* has occurred.

Death ultimately results from exhaustion, and is frequently preceded by the supervention of disseminated tuberculosis, giving rise to the febrile and toxemic symptoms of that condition. Occasionally a large hemorrhage brings about a fatal issue by suffocation, from inspiration of a portion of the fluid.

Pulmonary tuberculosis is not rarely complicated by or associated with other tuberculous affections, especially laryngeal tuberculosis and intestinal tuberculosis, the symptoms of which are then superadded.

What are the physical signs of the first stage of pulmonary tuberculosis?

The *chest* may be full and symmetrical. The *respiratory excursion* may be sufficient; perhaps, by standing behind the patient and placing the hands on the front of his chest, a slight deficiency of expansion at an apex may be detected. The *percussion-resonance* is vesicular, except over a small area at the uppermost portion of one lung, where there is slight relative impairment. At this point, too, the vesicular murmur is altered. The inspiratory murmur is less soft and breezy than normal, while expiration is prolonged and heard more distinctly than usual; or inspiration is jerking and arhythmical. Careful *auscultation* by an acute observer may detect fine moist râles at the end of inspiration. *Vocal resonance* and *fremitus* are slightly increased. If there is any expectoration, careful search may detect a small number of tubercle-bacilli in the *sputum*.

What are the physical signs of the second stage of pulmonary tuberculosis?

The *respiratory frequency* is slightly accelerated. The upper part of the chest is less full on one side than on the other, and the *expansion* in this area is deficient. The *resonance* here is impaired; the *percussion-note* may be dull. The vesicular element of the respiratory murmur is wanting. Inspiration is harsh; expiration is prolonged and blowing; the breathing may be bronchial. Fine crackling sounds are heard. *Vocal resonance* and *fremitus* are notably increased. Blowing sounds may be heard on stethoscopic auscultation over the subclavian vessels. The *sputum* contains many bacilli.

What are the physical signs of the third stage of pulmonary tuberculosis?

The general emaciation is striking. Decided depressions exist above and below the clavicle, on one or on both sides. The ribs are prominent, the interspaces narrowed. The *chest* appears rigid; it may heave, but it scarcely expands in respiration. The *percussion-dulness* is marked, perhaps in different degree over the upper lobes of the two sides. Here and there, in irregular areas, are heard the dull sounds of a thickened pleura. In the midst of the dulness a tympanitic or amphoric or cracked-pot sound is elicited. Coarse and fine râles, gurgling, bronchial or cavernous breathing are heard. Bronchophony or whispering pectoriloquy are present. *Tactile fremitus* is increased. The *sputum* contains bacilli and elastic fibers.

What are the best methods for detecting the presence of tubercle-bacilli in sputum?

In all bacteriologic investigations, the instruments and appliances used must be rigidly sterile. Sputa for examination should be collected in glass or porcelain vessels that have been made clean by boiling, and finally washed with a 1 : 1000 solution of mercuric chloride. The examination should not be too long deferred. In the interval the vessel should be covered.

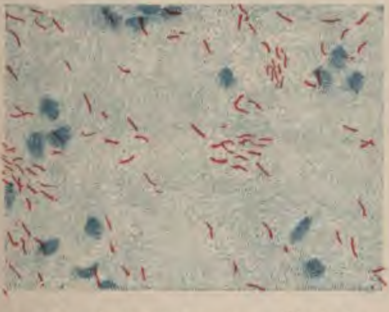
The sputum should be poured from its receptacle on a sterilized plate of glass having a black background. With a platinum wire, first heated to redness in the flame of a lamp or

Bunsen burner and permitted to cool, a small quantity of purulent matter is taken up and spread in a thin layer upon a cover-glass that has been washed with alcohol, ether, and bichloride solution and carefully wiped dry. The thin layer of sputum is permitted to dry at ordinary temperature, or the process is accelerated by gentle heat. When perfectly dry, the cover-glass preparation is drawn three or four times through the flame so as to fix the albuminoid matters.

The preparation is now ready to be stained. For the staining, a filtered solution composed of one part of fuchsin (or gentian violet), four of carbolic acid, ten of alcohol and one hundred of sterilized distilled water, may be used ; or ten or fifteen drops of anilin oil and about a dram of sterilized distilled water are shaken together and filtered, and then sufficient of a saturated alcoholic solution of gentian-violet (or fuchsin) addded until a deep coloration is produced.

The cover-glass preparation may now be floated on the surface of one of the solutions indicated, for twenty-four hours, at the ordinary temperature, or the process may be accomplished in fifteen minutes by the application of moderate heat until the vapor of steam arises ; or face upward, a few drops of either of the solutions indicated are placed upon the surface of the cover-glass preparation, which is gently heated until the vapors of steam arise.

FIG. 23.



Tubercle-bacilli in sputum. (Ziegler.)

The excess of free stain is removed with water. The preparation is decolorized in a solution of nitric acid (1 : 3), or in one of sulphuric acid (1 : 4). It is then briefly passed through seventy per cent. alcohol. It may at once be dried and mounted and examined, but it is better to stain again with an aqueous solution of a color complementary to that first used—fuchsin and methylene-blue, gentian-violet and vesuvin or Bismarck-brown.

The search for bacilli in sputum may be facilitated by adding to about four ounces of sputum, one ounce of sterilized distilled water, and from four to eight drops of liquor sodæ and heating; from two to three ounces more of water are added and the mixture is put aside in a conical glass for from twenty-four to forty-eight hours, when the supernatant fluid is decanted and cover-glass preparations made from the sediment.

What is the clinical course of "galloping consumption"?

The rapid form of confluent pulmonary tuberculosis, known as "quick consumption" or *phthisis florida*, as distinguished from the infectious fever known as acute miliary tuberculosis, not infrequently supervenes upon a neglected "cold," commonly a catarrhal pneumonia of the apex; or it may suddenly become manifest after localized tuberculosis of the apex, so slight as to be unsuspected, has existed for a longer or shorter time; it may closely or remotely follow an attack of influenza; or its immediate antecedents may be untraceable. Frequently, the subjects of phthisis florida present an hereditary liability to tuberculosis.

Clinically the disease is marked by high temperature of a continued or, more frequently, a remittent type; profuse night-sweats; profound digestive and circulatory disturbance; rapid wasting, with accompanying weakness; and pulmonary symptoms of progressive severity, with corresponding physical signs. Pathologically it is characterized by rapid caseation, with formation of extensive cavities; severe laryngeal and intestinal complications are frequent. It may apparently begin as an acute laryngitis of ordinary or of tuberculous type. Sometimes persistent diarrhea is the earliest symptom to attract attention.

The duration of the disease is from a few weeks to about eighteen months.

How is a pulmonary hemorrhage to be discriminated from a gastric hemorrhage?

The blood of pulmonary hemorrhage has a sweetish or saltish taste, is usually fluid, bright-red, and more or less frothy. In gastric hemorrhage the blood is usually dark and clotted; its taste is masked as a result of admixture with the contents of the stomach; the stools that follow are tarry.

Pulmonary hemorrhage may occur with or without cough, and is not provoked by taking food. Gastric hemorrhage usually occurs with vomiting, and is provoked by taking food.

How is the distinction to be made between pulmonary tuberculosis and chronic pleurisy?

Pleural adhesion and thickening are extremely common. They occasion retraction of the chest-wall, often more or less displacement of the heart, and give rise to dulness on percussion, enfeebled transmission of the breath-sounds, and increased vocal resonance and fremitus; but they are not associated with the auscultatory and constitutional phenomena of pulmonary tuberculosis, or with the presence of tubercle-bacilli in the sputum. If a pleural effusion is present, there are bulging of the lower part of the chest, flatness on percussion, feebleness or absence of breath-sounds, and diminished vocal resonance and fremitus. If the fluid is purulent, there are chills, fever, sweats and emaciation.

How is pulmonary carcinoma to be distinguished from pulmonary tuberculosis?

Carcinoma of the lung is a secondary affection. The metastatic growths give rise to irregularly distributed, multiple areas of percussion-dulness, without corresponding change in the auscultatory phenomena. The temperature is likely to be sub-normal rather than febrile, unless inflammation of the lung or pleura is excited. The duration of pulmonary carcinoma is limited; pulmonary tuberculosis may be indefinitely protracted. Tubercle-bacilli are not found in the sputum in pulmonary carcinoma. The presence of carcinomatous new-growths elsewhere is significant in doubtful cases.

How is pulmonary syphilis to be distinguished from pulmonary tuberculosis?

Syphilis of the lung appears either in the form of gummata or as fibroid induration. It is to be distinguished from tuberculosis by the absence of bacilli from the sputum, by the involvement of the middle and lower parts rather than the upper part of the lung, by the diffuse and irregular rather than concentrated localization of physical signs, by the absence of the constitutional phenomena of tuberculosis, and by the history or the knowledge of the existence of other syphilitic manifestations. The therapeutic test is sometimes available, but should be cautiously applied, as cases of tuberculosis are usually injured by potassium iodide.

Syphilis and tuberculosis may coëxist.

How are bronchiectasis and pulmonary tuberculosis to be differentiated?

Bronchiectasis is attended with copious muco-purulent expectoration, and perhaps with decided emaciation; the pulmonary resonance may be impaired, and large, moist râles may be heard; but the physical phenomena are usually bilateral in distribution and most decided at the bases of the lungs; pulmonary tuberculosis usually begins at the apex of one lung, whence it extends. Bronchiectasis is slowly if at all progressive; tuberculosis less commonly stops short of a fatal termination. The sputum of a case of bronchiectasis does not contain tubercle-bacilli.

How is an abscess of the lung to be diagnosticated from pulmonary tuberculosis?

An abscess of the lung may develop in the course of a pneumonia or an empyema, as a result of traumatism, or it may constitute a manifestation of a general pyemia. It is to be distinguished from tuberculosis by a knowledge of its possible origin, by the appearance of the phenomena in a lower rather than in an upper lobe and upon one side only. The condition is acute rather than chronic, and the sputum does not contain tubercle-bacilli. The physical phenomena are peculiarly circumscribed.

By what means are malarial fever and pulmonary tuberculosis to be differentiated?

When caseation and suppuration are taking place in a lung, the temperature is high in the evening, declining towards morning. In addition, however, there are persistent dyspnea, increased frequency of breathing, cough, expectoration containing tubercle-bacilli, loss of flesh, night-sweats, dulness on percussion, and mucous râles on auscultation. The symptoms last enumerated are wanting in intermittent fever, while the plasmodia of malaria are wanting in the blood in pulmonary tuberculosis. Possible mistakes in diagnosis are to be avoided by a physical examination.

How does tuberculosis of the bronchial glands differ from whooping-cough?

Whooping-cough is a self-limited disease; tuberculosis is not. Grave constitutional symptoms attend the latter; recovery from whooping-cough is comparatively rapid. While the cough induced by tuberculosis of the bronchial glands is ringing, it does not possess the peculiar character of the cough of pertussis. Cases of whooping-cough do not occur isolated. By pressure on adjacent structures, enlarged bronchial glands may occasion dyspnea, cough and cyanosis, and edema of the face and neck.

Acute Miliary Tuberculosis.

What are the symptoms of acute miliary tuberculosis?

Acute miliary tuberculosis sets in insidiously. The victim may be a member of a tuberculous family. He is taken ill with slight cough, scanty or no expectoration, marked dyspnea, elevation of temperature, rapidity of pulse, delirium, and other symptoms of a grave intoxication. Tuberculous involvement of the bowel will cause diarrhea; of the cerebral meninges, the signs of meningitis. The course of the disease may be rapid or protracted. Death may take place from exhaustion, or, in rarer instances, the acute symptoms subside, and those of chronic tuberculosis appear.

What are the physical signs of acute miliary tuberculosis?

The physical signs of acute miliary tuberculosis are at first practically those of an acute bronchitis: a full chest; rapid breathing; unaltered percussion-resonance; harsh, vesiculo-bronchial respiratory sounds; unchanged vocal resonance and fremitus. Later, dulness at one apex or at both apices, or just below, and fine crackling râles are heard; and still later, evidences of caseation are found on auscultation and percussion. The detection of tubercle-bacilli may be very late. Some cases early develop indications of apex-pneumonia.

How are acute miliary tuberculosis and typhoid fever to be distinguished from one another?

The prostration and the general condition are much alike in both; but in acute miliary tuberculosis the epistaxis, the rose-spots, the characteristic temperature-course, and the peculiar stools of typhoid fever are wanting. The temperature of acute miliary tuberculosis presents considerable oscillations on the same day; without therapeutic interference, it may be normal, subnormal and very high. Bronchitis may attend typhoid fever, but the dyspnea is never so marked as it is in acute miliary tuberculosis. The progress of the case determines the diagnosis. Not only do the marked physical signs of destructive change in the lungs develop in the one disease, and not in the other, but typhoid fever is a self-limited disease of known duration, with a tendency to recovery, while acute miliary tuberculosis is a disease of uncertain duration, and sooner or later almost invariably fatal.

Tubercle-bacilli may sometimes be discovered in the sputum of acute miliary tuberculosis.

The knowledge of previous scrofulous or tuberculous disease in the patient, or a family history of scrofula or tuberculosis, should excite suspicion of acute tuberculosis; and in susceptible subjects the latter disease may even quickly follow typhoid fever.

Interstitial Pneumonitis.

What are the characteristics of interstitial pneumonitis?

As a result of the irritation occasioned by the constant inhalation of fine particles of dust by miners and grinders, and others, a chronic bronchitis with hyperplasia of the interstitial pulmonary tissue develops. The two layers of pleura are often adherent and thickened. As time progresses, contraction takes place, terminating in condensation of the lung and dilatation of the bronchial tubes. Interstitial pneumonitis may develop as a part of a general fibroid degeneration.

In coal miners the condition is known as *anthracosis;* in iron-workers as *siderosis;* in grinders as *chalicosis.*

What are the symptoms of interstitial pneumonitis?

There is obstinate cough and abundant muco-purulent expectoration, which sometimes contains particles of the dust inhaled. There may be considerable wasting and dyspnea.

What are the physical signs of interstitial pneumonitis?

The *chest* is diminished in size, rather flattened anteriorly, from retraction. The *respiratory excursion* is small, the chest expanding but little in inspiration. The *percussion-resonance* is impaired over a large area of pulmonary surface. The *breathing* is bronchial. Large moist and dry râles are heard. The *vocal resonance* and *fremitus* are increased.

How does interstitial pneumonitis differ from chronic pleurisy?

Interstitial pneumonitis and chronic adhesive pleurisy are allied conditions and are often associated. Occurring alone the former is wider in its distribution and is more likely than the latter to be bilateral. Blowing breathing, râles, cough and expectoration attend the pulmonary, but not the pleural, condition.

Pulmonary Emphysema.

What are the characteristics of pulmonary emphysema?

Pulmonary emphysema is usually a result of long-continued excessive expiratory effort with closed glottis—as in protracted cough or in the habitual blowing of wind instruments. It is often associated with chronic bronchitis. The air-vesicles become dilated into large sacs, and the bloodvessels in the interlobular septa are obliterated. The increased work thrown upon the right heart in turn gives rise to dilatation.

Emphysema manifests itself by dyspnea, expiratory in character; shortness of breath, aggravated in paroxysms, and attended with distressing cough and scanty expectoration. The cardiac insufficiency adds to the dyspnea and may be the cause of dropsy.

What are the physical signs of emphysema?

The *chest* is large, "barrel-shaped;" the circumference being increased in greater degree than the vertical diameter. The *respiratory excursion* is slight and may be scarcely visible; although the chest may rise and fall as a whole, the ribs being fixed in the position of full inspiration. The *percussion-resonance* is heightened, almost tympanitic. The *breath-sounds* are feeble, being almost continuous and without intermission, expiration being prolonged and attended with a succession of puffs. The *vocal resonance* and *fremitus* are diminished. The area of superficial cardiac dulness is diminished, but deep percussion will reveal enlargement of the right ventricle.

How is emphysema to be distinguished from pneumothorax?

Pneumothorax usually results from the breaking through the pleura of a destructive process in the lung, such as the caseation and suppuration of tuberculosis. The symptoms to which it gives rise are abrupt in onset and distressing in character. The patient perceives a sudden, severe pain in the side, and is seized with great dyspnea and a sense of impending death. The chest is seen to be enlarged, the breathing rapid and shallow, the respiratory excursion small; the percussion-resonance is tympanitic and the breath-sounds are feeble. In pneumothorax, the dilatation of the chest and the tympany are, however, unilateral; in emphysema, they are symmetrical. In pneumothorax, an amphoric blowing sound is heard on inspiration, and if pleurisy with effusion have set in, metallic tinkling, succussion sounds and egophony as well. The recognition of a condition that may give rise to perforation and pneumothorax aids in the diagnosis.

How is emphysema to be distinguished from a pleural effusion?

An effusion into the pleura may take place in the course of a pleurisy, or as a result of disease of the heart or kidney. Adjacent organs are displaced in proportion to the quantity of fluid poured out. If the percussion-note be hyper-resonant at the upper part of the chest, above the level of the fluid, it is flat below. Pleuritic effusions are usually unilateral; when the effusion is part of a general dropsy it is usually bilateral. Emphysema is rarely unilateral. In emphysema, the breath-sounds are everywhere feebly heard; in pleural effusion the breathing may be puerile, or even bronchial, above the level of the fluid, but the breath-sounds are not well heard through the fluid. Vocal resonance and fremitus are diminished in emphysema; absent below the level of a pleural effusion. If the effusion becomes purulent, rigors, hectic fever, sweats and marked emaciation occur.

Pneumothorax.

What are the clinical features of pneumothorax?

Pneumothorax may result from traumatism that causes fracture of a rib and perforation of the lung; from the rupture of an emphysematous pulmonary alveolus; from communication between the pleural cavity and an adjacent hollow viscus; from the softening of a septic infarct or of an area of pneumonic consolidation; but the most common cause is ulcerative perforation of a tuberculous cavity in the lung. The phenomenon usually *sets in* suddenly, with sharp pain in the side, intense dyspnea and a sense of great oppression. The *breathing* is hurried and shallow. The *chest* on the affected side becomes bulging; the *percussion-note* is tympanitic; the *breath-sounds* are enfeebled or distant, perhaps amphoric; as fluid is poured out, metallic tinkling may be heard, and by shaking the patient, with the ear applied to his chest, a succussion-sound may be elicited; the *voice-sounds* and *vibrations* are poorly transmitted. The *heart*, as well as other viscera, may be displaced.

How are pneumothorax and diaphragmatic hernia to be differentiated?

The stomach may be dragged upwards beneath the ribs by a contracting lung, or the stomach or the large intestine may bulge through a yielding portion of the diaphragm, so that there may be unilateral percussion-tympany, with displacement of the heart; vibrations of the fluid in the hollow viscus may give rise to metallic tinkling, which, however, occurs independently of respiration and is associated with rumbling sounds peculiar to the gastro-intestinal tract. The interference with the respiratory functions that results is, moreover, not only acute in onset, as it is in pneumothorax (of which the etiologic elements are wanting), but it may be equally sudden in disappearance. Diaphragmatic hernia may have a history dating from birth; it bodes no danger unless strangulation occurs, of which the symptoms are characteristic. The outcome of pneumothorax is in the nature of things often rapidly fatal.

How are pneumothorax and a pulmonary cavity to be differentiated?

A large cavity in the lung may yield a tympanitic percussion-sound, amphoric respiration, metallic râles or tinkling, and succussion-sounds, but the phenomena are usually circumscribed; there is no bulging, no acute exacerbation of pain, dyspnea and oppression, and the percussion-flatness of an effusion into the pleural sac is wanting.

How is pneumothorax to be distinguished from a pleural effusion?

Pneumothorax sets in suddenly, with acute pain and dyspnea. A pleural effusion usually takes place insidiously. Pleurisy with effusion usually attends pneumothorax, so that the signs of both may be present; but when the pneumothorax is the primary condition, the percussion-note above the level of the effusion is more pronouncedly tympanitic and the voice more distinctly egophonic. Succussion-phenomena and tinkling are not heard unless both air and fluid are present in the pleural sac. Finally, the recognition of a condition that gives rise to pneumothorax may decide the diagnosis.

Asthma.

What are the symptoms of asthma?

Asthma is a paroxysmal affection, the symptoms of which are probably dependent upon spasmodic narrowing of the smaller bronchial tubes. The attacks recur at irregular intervals, with or without apparent exciting cause. Among provocative conditions are changes of climate and weather, overeating, indigestion, emanations from feathers and sexual excitement.

The seizure is sudden in *onset*, usually occurring at night. The patient is awakened with a sense of oppression and distressing dyspnea, inspiratory in character. Orthopnea is common. During the *paroxysm*, which may last for several hours, the chest heaves spasmodically, but the *pulmonary expansion* is slight; the *face* is pale or livid, and the distress is evident. Loud, wheezing sounds are heard. On auscultation, the *breath-sounds* are feeble, or the vesicular murmur may be obscured by the wheezing or replaced by sonorous and sibilant râles. The *percutory phenomena* are unaltered. The *crisis* takes place with a profuse expectoration of mucus, perhaps, also, with a copious discharge of limpid urine. This may terminate the attack, or the same phenomena may be repeated on several successive nights or even invade the day.

Asthma may be primary or secondary. It may result from reflex influences, such as disease of the nose. It may apparently replace the convulsive seizure of epilepsy. Asthmatoid seizures are common accompaniments of chronic bronchitis and emphysema, of cardiac insufficiency, and of chronic nephritis.

How is asthma to be distinguished from an asthmatoid condition?

In true asthma, no cause for the disease may be found or else some local or reflex irritation may be discovered. When so-called asthmatoid attacks occur, their discrimination depends upon the recognition of an organic pulmonary affection, such as emphysema or bronchitis, or of cardiac incompetency or of chronic nephritis. Such attacks do not pursue the typical course of true asthmatic seizures and do not terminate suddenly with profuse expectoration.

How does asthma differ from whooping-cough?

Whooping-cough is a disease of children; asthma, a disease of adults. The characteristic whoop of pertussis is wanting in asthma. An attack of whooping-cough does not last more than a couple of months, at most; asthma may continue indefinitely.

What are the points of differentiation between asthma and paralysis of the diaphragm?

The dyspnea resulting from paralysis of the diaphragm is not paroxysmal, as is that of asthma. Paralysis of the diaphragm is not characterized by an absence of the breath-sounds, followed by wheezing and high-pitched râles. When the diaphragm is paralyzed, inspiration is attended with expansion of the chest and depression of the abdominal wall; in expiration the chest collapses and the abdominal wall is elevated; stimulation of the phrenic nerves restores the normal harmony of action; attempts at bearing down are futile or ineffective.

THE DIGESTIVE SYSTEM—THE MOUTH.

Catarrhal Stomatitis.

What are the clinical characteristics of catarrhal stomatitis?

Catarrhal inflammation of the mucous membrane of the mouth may *result* from the ingestion of irritating substances, from the presence of carious teeth, or by extension from adjacent disease; it may also develop in conjunction with morbid dentition, or derangement of digestion, or in the course of the exanthemata. It *manifests* itself by redness, tumidity, and increased heat of the structures within the mouth, and by increased secretion. The taking of food is attended with discomfort or with pain; taste is impaired; and the breath is offensive. As a rule, there is little or no constitutional disturbance.

Aphthous Stomatitis.

What are the clinical features of aphthous stomatitis?

In individuals exposed to unfavorable hygienic conditions, and in those debilitated by disease, small discrete or confluent, grayish or whitish masses of degenerated epithelium accumulate at various parts of the mucous membrane of the mouth; the desquamation of the masses exposes subjacent ulceration. Mastication in adults and nursing in infants are difficult and painful; the secretions of the mouth are increased; the appetite may be impaired; digestion may be deranged; and there may be diarrhea.

Thrush.

What are the clinical manifestations of thrush?

Thrush, *muguet* or *parasitic stomatitis* is a mycotic inflammation of the mucous membrane of the mouth and throat, to which children are especially prone. It is dependent upon the presence of a fungus, termed the *mycoderma* (or *oïdium*) *albicans*. An acid reaction of the secretions of the mouth is an essential condition for the development of the affection. The growth of the fungus and the resulting irritation give rise to the formation of minute curd-like masses upon various parts of the mucous membrane. Forcible detachment of the masses occasions bleeding. The *symptoms* are those of the ordinary form of stomatitis, plus the flaky deposits containing the characteristic fungus. There may also be diarrhea and a varying degree of constitutional disturbance. In nurslings, the inability to suckle may result in inanition and death.

Ulcerative Stomatitis.

What are the symptoms of ulcerative stomatitis?

Ulcerative stomatitis is an aggravated form of inflammation of the mouth, attended with ulceration, which it is stated is usually unilateral. The affection *arises* amid conditions of crowding and filth, and in those supplied with insufficient and inappropriate food; it may also be a sequel of other forms of stomatitis, of caries of the teeth, or of the maxillary bones. It is *manifested* by impaired appetite, fetid breath, increased salivation, pain in eating, and constitutional symptoms of varying intensity. Sometimes adjacent lymphatic glands undergo enlargement.

Mercurial Stomatitis.

What are the clinical manifestations of mercurial stomatitis?

Stomatitis sometimes *results* from the medicinal ingestion of large quantities of *mercury*, or of small quantities by persons possessing an idiosyncrasy, or as a manifestation of mercurial intoxication by means of articles of food or drink, or from exposure to the metal in certain occupations. The *symptoms* vary greatly in severity. The gums especially become swollen, reddened, tender, and sometimes ulcerated. The *teeth* may fall out, and the maxillary bones become carious. The *breath* is fetid. The *saliva* contains mercury; its secretion and discharge are inordinately increased, giving rise to the term "salivation," as descriptive of the affection.

Gangrenous Stomatitis—Noma.

What are the clinical features of gangrenous stomatitis?

Gangrenous stomatitis, *noma* or *cancrum oris* is essentially an affection of childhood, rare, and almost invariably fatal, which *develops* in those of depraved constitution, often at the termination of one of the exanthemata, particularly measles. The disorder is *manifested* by a brawny induration of one cheek, the structures of which undergo disintegration, with resulting ulceration of the mucous and cutaneous surfaces, and, not rarely, perforation. Adjacent portions of the gums may by contiguity become involved in the process. The teeth may fall out and the maxillary bones become carious. The early symptoms may be obscured by those of the antecedent condition. Soon, however, the breath becomes fetid, and, in addition to the local manifestations, the symptoms of septic intoxication may appear, in the midst of which the child may die. *Recovery* may take place, with hideous deformity of the face. Pneumonia, pulmonary gangrene, and entero-colitis may be *complications*.

THE TONGUE.

Glossitis.

What are the symptoms of glossitis?

When the tongue is inflamed, from whatever cause, the organ becomes enlarged, tumid, reddened, painful; speech, deglutition and mastication, sometimes respiration, are interfered with, and the secretions of the mouth are increased. The swelling may be so great that suffocation results, unless relief be given by incision. Glossitis may be *superficial* or *parenchymatous*, *acute* or *chronic*; the intensity and character of the symptoms being modified accordingly. It may be due to erysipelas, either primarily or secondarily. Epidemics of erysipelatous glossitis have occurred, and the name "black tongue" has been applied to this affection. Parenchymatous glossitis sometimes proceeds to suppuration.

Leukoplakia Lingualis.

What is leukoplakia of the tongue?

Leukoplakia lingualis, or *leukoplakia buccalis*, is a name applied to a peculiar chronic affection of the tongue or of the tongue and buccal mucous membrane, characterized by the formation of persistent, horny, whitish patches upon the surface, sometimes extending entirely through the epithelial layer. Untreated, it is said to lead at times to the development of carcinoma. Sometimes it appears to be related with gout.

Glossanthrax.

What is glossanthrax?

Glossanthrax is a term applied to the localization of malignant pustule upon the tongue. It is to be differentiated from carcinoma, tuberculosis, syphilis and other affections leading to suppuration or ulceration. The local and constitutional symptoms are those of anthrax in general. The appearance of the eschar is characteristic and the presence of anthrax-bacilli is diagnostic.

Nigrities.

What is nigrities?

Nigrities, also called *black tongue* and *hairy tongue*, is an affection of the filiform papillæ of the tongue, supposed to be due to the irritation of a special fungus. The papillæ in various situations become discolored, thickened, and elongated, giving the appearance of a scattered or compact hairy growth upon the dorsum of the tongue. Desquamation takes place, after which the tongue may remain comparatively clean for a longer or shorter period; then the growth recurs. The affection is to be differentiated from staining of the tongue by tobacco, medicines, and the like.

MUMPS—PAROTIDITIS.

What are the symptoms of parotiditis?

Parotiditis or *mumps* may be primary or secondary in the course of infectious diseases. It is *characterized* by pain at the angle of the jaw, followed by tumefaction of the parotid gland, at first on one side and then on the other. Movement of the jaw, as in mastication, is difficult and painful. Deglutition is not interfered with; hearing may be deranged. The secretion of saliva is usually excessive. There are febrile manifestations of moderate severity.

Orchitis or ovaritis is a peculiar *complication* of parotiditis. It is likely to occur as the parotid swelling subsides.

THE PHARYNX.

Pharyngitis.

What are the symptoms of pharyngitis?

In *acute catarrhal pharyngitis*, or *angina*, the symptoms vary with the intensity and extent of involvement. Ordinarily, there are "sore throat," irritable cough, pain or difficulty in deglutition, interference with respiration, enlargement of the tonsils and of the glands of the neck. On *inspection*, the soft palate, the uvula, the tonsils, the posterior and lateral walls of the pharynx, or the palatine arches may, one or more, be seen to be reddened and tumefied, and often coated with glairy mucus. A moderate degree of *fever* attends acute pharyngitis.

Acute phlegmonous pharyngitis is a much more serious affection, involving not only the mucous membrane, but also the submucous connective tissues and even at times the sheaths of the muscles. The constitutional symptoms are in accordance with the severity of the process, and may be those of pyemia. The pus may gravitate to the cellular tissues of the neck, manifesting as an external swelling and causing dyspnea or even suffocation from compression of the trachea. The inflamed tissues of the throat, especially the soft palate and uvula, may be greatly swollen and edematous.

What is acute tuberculous pharyngitis?

Acute tuberculous pharyngitis is a form of acute miliary tuberculosis, apparently beginning in the pharynx.

The *constitutional manifestations* are those of an acute febrile process of grave type, sometimes simulating typhoid fever.

Locally, deposits of tubercle may be observed beneath the mucous membrane as little semi-transparent, grayish nodules, resembling in size and form vermicelli-seeds or fish-eggs. These are collected into little patches more or less confluent, which eventually undergo ulceration.

The uvula is sometimes thickened into a somewhat characteristic gelatinous-looking, reddened, club-shaped mass. This gelatinous thickening may likewise take place in other portions of the pharynx.

The disease may extend to the epiglottis, tongue, and larynx, or to the vault of the pharynx and the nasal passages.

The ulcerative process usually begins on a palatine fold or on a lateral wall of the pharynx, whence it rapidly extends. Ulceration may penetrate the submucous tissues, and the muscles may undergo tuberculous or fatty degeneration. Pus is usually absent from the surface of the ulcers and the bacillus tuberculosis is sometimes found in the detritus.

The chief and most distinctive *local subjective symptom* is intense pain in swallowing, often more than can be accounted for by the extent of visible disease. The pain may extend into the ears.

As the disease progresses, cough is superadded, emaciation becomes rapid, and signs of pulmonary disease, and perhaps of other complications, become manifest.

Death may result within a few weeks, and is rarely postponed beyond two or three months.

Recovery is the exception.

How is tuberculous pharyngitis to be distinguished from syphilitic sore-throat?

The intense pain in swallowing, the characteristic deposit and gelatinous infiltration, the absence of pus from the ulcers, the history of the attack, the personal history, the family history of the patient, and the febrile symptoms; the discovery of the tubercle-bacillus in the detritus of the ulcers, or in the sputum, together with the detection of the evidence of pulmonary tuberculosis as the case proceeds, are the points upon which the diagnosis from syphilis must depend.

Syphilitic and tuberculous disease may, however, coëxist.

The supervention of typhoid symptoms in a case of supposed syphilitic ulceration of the throat should excite suspicion of the existence of tuberculosis.

How is tuberculous pharyngitis to be distinguished from typhoid fever?

Typhoid fever is sometimes accompanied by ulcerative pharyngitis and laryngitis, though the cases in which this occurs are much rarer in North America than they appear to be in Europe. The characteristic fish-egg-looking infiltration of tuberculosis and the non-purulent character of the ulceration would make the discrimination locally; while, constitutionally, there would be absence of the characteristic temperature-course, of the rose-spots, and of the peculiar stools of typhoid fever. Discovery of the tubercle-bacillus would be conclusive.

Tonsillitis.

What are the symptoms of tonsillitis?

When the tonsils are inflamed, adjacent parts usually participate in the process. *Parenchymatous tonsillitis* or *quinsy* may be primary or secondary to various infectious diseases. It sometimes *sets in* suddenly, with a chill, followed by decided elevation of temperature and other febrile manifestations. Usually, one tonsil only is affected, or first one and then the other, but bilateral involvement may occur and the glands become so intensely swollen as to meet in the middle line, practically obstructing deglutition and respiration. In such cases the voice is nasal, and fluids attempted to be swallowed may return through the nose. There is always some interference with swallowing. The pain is atrocious, and may extend into the ear on the affected side. There is increased secretion of saliva; swallowing is increased in frequency, and aggravates the pain. Sometimes the patient lies with open mouth, making labored and noisy efforts at respiration, while the saliva dribbles. On examination, the tonsils are seen to be enlarged and angry, adjacent parts in some degree participating in the inflammatory process.

The enlargement of the glands can, sometimes, be distinctly detected from without. Tenderness on pressure beneath the angle of the jaw is common.

An inflamed tonsil or the peritonsillar tissues may suppurate, and grave complications, such as ulceration into the carotid artery, have occurred.

Lacunal or *follicular tonsillitis* is a much less serious affection. The process is superficial, the lining membrane of the lacunæ or ducts being involved rather than the substance of the gland. Scattered over the surface of the inflamed and enlarged tonsils are a number of yellowish points or patches, indicative of accumulations of sebaceous matter, desquamated epithelium and fungi, at the orifices of the ducts. These plugs may be readily removed by means of a scoop, sometimes by syringing—a point of some importance in the discrimination from diphtheria. Parenchymatous tonsillitis sometimes follows lacunal tonsillitis, and tonsillar or peritonsillar abscess may then develop. Untreated, the duration of tonsillitis is from two to ten days or more.

Some cases of tonsillitis are associated with pain and other rheumatic manifestations in the muscles or joints, and endocarditis and pericarditis have been noted. An endocardial murmur is not uncommon. Swelling of the joints may appear in apparent metastasis as a tonsillar inflammation declines.

Anomalous eruptions and albuminuria are among the less common concomitants of tonsillitis. Paralyses are rare sequelæ.

Herpetic Sore-Throat.

What is herpetic sore-throat?

Herpetic sore-throat, *herpetic tonsillitis*, *herpes of the pharynx*, *common membranous sore-throat*, *ulcero-membranous angina*, *diphtheroid throat*, are names applied to a disease often mistaken for diphtheria, but which is, in reality, a form of inflammation of the mucous membrane of the palate, tonsils, uvula and pharynx, characterized by the eruption of herpetic vesicles, which soon rupture, leaving little circular ulcers that coalesce and become covered with a fibrinous exudation. It is sometimes associated with herpes of the lips.

Constitutional symptoms may be absent, but, when present, are usually of a mild febrile type. There may, however, be high fever, preceded by malaise or chill.

The pain in deglutition (odynphagia), and the dryness and heat of the throat are often much greater than in ordinary forms of pharyngitis. In rare instances, in children, the false membrane has extended into the larynx, causing suffocation. Usually the disease terminates in recovery in about a week or ten days.

Chronic or *recurrent herpes of the throat* is encountered in rare instances.

How is common membranous sore-throat to be distinguished from diphtheria?

The diagnosis is sometimes very difficult, and, when in doubt, the safer plan is to consider the case one of diphtheria. As a rule, however, the islet-like distribution of the patches of fibrinous exudation covering the ulcers left by rupture of the vesicles of herpes is quite different from the appearance presented by the coherent, continuous mass of thick, yellowish or grayish membrane observed in diphtheria. Herpes is more frequent upon the palate and tonsils. Diphtheria usually involves the pharynx extensively. The constitutional symptoms of diphtheria are, as a rule, much more profound than those of herpetic sore-throat. Herpes of the lips often coëxists with herpetic sore-throat, very rarely with diphtheria. Diphtheria is contagious. Herpes is non-contagious.

Gangrenous Pharyngitis.

What is gangrenous pharyngitis?

Gangrenous pharyngitis, or *putrid sore-throat*, may *originate* independently of any other malady or may follow ordinary forms of pharyngitis, or the sore-throat of the exanthemata, or of dysentery, or of typhus or of typhoid fever. It sometimes occurs in cases of tuberculosis. *Constitutional symptoms* are typhoid in type. The *local symptoms* are those of violent inflammation of the mucous membrane of the tonsils, palatine folds, and walls of the pharynx, which soon become covered with gangrenous patches. The destructive process rapidly extends, sometimes into the esophagus, the larynx, and the nares. Sometimes the process is extremely limited, as to the tonsils. Erosion of blood-vessels may cause fatal hemorrhage.

How is gangrenous sore-throat to be distinguished from diphtheria?

In gangrene, the patches are grayish-black in color from the outset, while the pseudo-membrane of diphtheria becomes dark only as the disease progresses.

Swelling of the cervical glands is unusual in putrid sore throat, and the characteristic odor of gangrene is almost unmistakable.

Retro-pharyngeal Abscess.

What are the symptoms of retro-pharyngeal abscess?

Suppuration in the retro-pharyngeal tissues is most commonly a result of destructive disease of the cervical vertebræ; it may also be due to inflammation of the lymphatic glands or of the connective tissues, resulting from traumatism or developed in the course of infectious disease, or by extension from adjacent disease. The affection is more common in children than in adults, and in tuberculous or syphilitic than in other subjects. There may be an initial chill, with nausea and vomiting, followed by considerable elevation of temperature and acceleration of pulse.

There are soreness of the throat, with pain and difficulty of swallowing; orthopnea; suffocative paroxysms; and noisy breathing; the voice is muffled or nasal. The head is often thrown back. There is little or no cough.

The neck may be swollen and tender to touch, especially behind the angle of the jaw, in front of the sterno-mastoid muscle. The submaxillary glands may suppurate and fluctuation become evident.

On *inspection* of the throat the posterior wall of the pharynx may appear tumid, or a distinctly circumscribed projection may be seen, the mucous membrane over and around the swelling being reddened, perhaps ecchymotic.

The abscess may be so hidden that the use of the mirror may be necessary for its detection.

On *palpation* fluctuation may be elicited.

THE ESOPHAGUS.

Stricture of the Esophagus.

What are the symptoms of stricture of the esophagus?

The esophagus may be narrowed by new-growths in its walls, by cicatrices resulting from previous syphilitic or tuberculous disease or from the ingestion of corrosive substances, by pressure from without, as by an aneurism or a new-growth, or as a congenital malformation. Deglutition is interfered with, so that it may be possible to swallow only liquids. If the narrowing is decided, a pouch forms above the seat of constriction, in which considerable quantities of food accumulate, to be periodically rejected. Esophageal growths, if favorably situated, can sometimes be detected by the methods of laryngoscopy. *Esophagoscopy* has not yet been sufficiently developed for diagnostic purposes. Conclusive evidence of the existence of a stricture of the esophagus is furnished by the resistance encountered in the introduction and withdrawal of a bulbous bougie; if aneurism be suspected, this means of exploration is not permissible.

How are functional and organic strictures of the esophagus to be differentiated?

Spasmodic contraction of the esophagus is apt to result from the presence of a foreign body, as a bougie, in the gullet. Spasm of the esophagus (*esophagismus*), in a degree sufficient to give rise to symptoms, sometimes occurs in hysterical persons. Under the conditions last named, food may be obstinately rejected and a considerable degree of emaciation result. The introduction of a bougie may be met with some resistance, which, however, slowly yields to gentle pressure. Careful observation will disclose the fact that not all the food taken is rejected. A powerful impression, judiciously made, may at once cause the disappearance of the symptoms, which occur, without discoverable cause, in a person with other hysterical attributes.

THE STOMACH.

How may the duration of digestion be tested?

An experimental meal, consisting of gruel, an ordinary piece of beefsteak, and white bread, is given; after which nothing is to be taken until the examination is concluded. Seven hours after eating, the stomach is to be washed out by siphonage with a soft-rubber esophageal catheter. If digestion is normal the washings should contain no remains of the food, or at most but a few particles.

What are the tests for free hydrochloric acid in the gastric contents?

Many tests have been proposed, none of which is absolutely free from fallacy. For practical purposes, Congo-red and phloroglucin-vanillin seem to be the most available.

1. Strips of filter-paper saturated with Congo-red become blue if dipped in fluid containing free hydrochloric acid; organic acids likewise discolor Congo-red, but do not produce the same blue tint.

2. One or two drops of a solution consisting of phloroglucin, two parts; vanillin, one part; absolute alcohol, thirty parts, are placed in a shallow, porcelain capsule, with an equal volume of the filtrate obtained from the stomach-contents, and gently heated. If free hydrochloric acid is present in a proportion of 0.05 or more per thousand, a bright-red deposit will form. If no hydrochloric acid be present, the deposit is brownish-red or brown. The presence of sulphuretted hydrogen is said to be the only source of error.

How is the solvent power of the gastric juice tested

By the administration, on an empty stomach or one hour after a trial-breakfast of tea and toast, of potassium iodide in rubber capsules fastened with fibrin, and examination of the saliva for iodine-reaction by means of starch-paper and the fumes of strong nitro-hydrochloric acid. Normally the reaction should be demonstrable in from six to eleven minutes after the ingestion of the capsules.

Gastralgia.

What is gastralgia or gastrodynia?

Gastric pain is usually symptomatic of inflammation, ulceration or neoplasm. It may, however, occur independently of recognizable structural disease, and is then considered a neurosis—*neuralgia of the stomach.* The pain of neuralgia is spasmodic, of a cutting character, usually rather brief in duration, shooting and shifting in seat, and occurs spontaneously or immediately after the ingestion of food. It may induce and be relieved by vomiting or eructation. It is relieved by heat or pressure. It is not accompanied by vomiting of blood or by the usual manifestations of disordered digestion. Other neurotic symptoms may coëxist.

Acute Gastritis.

What are the symptoms of acute gastritis?

Acute gastritis results from the entrance of irritating matters into the stomach. It varies in degree in accordance with the intensity of the causative irritant. The milder attacks are sometimes called "*acute gastric catarrh*" or "*acute indigestion.*" Acute gastric catarrh may complicate infectious diseases, or its exciting cause may escape detection.

The *symptoms* are anorexia, nausea, vomiting, often epigastric pain and tenderness; in a severe attack, the *face* is pale and anxious, the *pulse* small and firm; the *skin* may be cold and clammy; there may be moderate elevation of *temperature;* syncope and collapse may occur. The vomited matters consist of the contents of the stomach, mucus, perhaps bile, and sometimes blood. There is often violent retching, without expulsion of the contents of the stomach, or occurring when the organ is empty. In addition there are commonly headache and thirst. The tongue may be red and angry. Perforation of the walls of the stomach may take place. Peritonitis may develop. The intestines are not likely to entirely escape.

How are the gastric symptoms occurring at the onset of acute febrile disorders or in the course of cerebral disease to be distinguished from the symptoms of acute gastritis?

The vomiting of acute, febrile disorders, or of cerebral disease, is not dependent upon the ingestion of food; nor is it necessarily attended with nausea, coated tongue, or pain in the epigastrium. Tenderness is not common. If symptoms indicative of a constitutional affection have not been obtrusive, careful investigation will succeed in detecting them at once, or after the lapse of a variable period. No single symptom, but an association of symptoms establishes the diagnosis.

How are acute gastritis and intestinal obstruction to be differentiated?

The vomiting of intestinal obstruction usually, but not invariably, becomes fecal; the rolling of the obstructed intestines may be apparent through the abdominal walls. The matter vomited in acute gastritis consists largely of mucus, perhaps of some blood, and shreds of mucous membrane; while there is a history of irritation, poisoning, or indiscretion in diet. Obstinate constipation attends obstruction; acute gastritis is rather likely to be associated with diarrhea. If an obstruction of the bowel be not speedily removed, death results. Recovery from the milder forms of acute gastritis may take place spontaneously.

Chronic Gastritis.

What are the symptoms of chronic gastritis?

Chronic gastritis or *chronic gastric catarrh* is the principal factor in the production of dyspepsia. The symptoms differ from those of acute gastritis not only in degree, but also in character. There are many varieties of the affection but the general phenomena are the same in all. The more or less persistent ingestion of food or drink, improper in quality or excessive in quantity, is the principal cause of chronic gastric catarrh. The disorder manifests itself by nausea, vomiting, impaired appetite, coated tongue, epigastric discomfort, aggravated by the ingestion of food, flatulence, eructations, pyrosis, acidity, bad taste, offensive breath, and palpitation of the heart. There is dull, diffuse abdominal pain, relieved by vomiting. There is also diffuse epigastric tenderness. The vomiting, as a rule, takes place from one-and-a-half to two hours after the ingestion of food. Constipation is common. Usually there is increased thirst. The complexion is often sallow. Headache is frequent and vertigo not uncommon. There may be loss of flesh and emaciation. Mental operations are in many cases temporarily sluggish, depression ("blues"), not rare, and sleeplessness or disturbed sleep and disquieting dreams quite common.

If much accumulation of undigested and decomposing food takes place, or impairment of the motor activity of the stomach becomes decided, dilatation of the viscus (*gastrectasia*) may result. A succession of meals may be taken before vomiting occurs; when it does, astounding quantities of liquid, containing the macerated and fermenting remains of food, and various fungi (especially the yeast-fungi and sarcinæ), are ejected. The quantity of hydrochloric acid in the gastric juice may or may not be changed. Organic acids of fermentation are usually present.

What are the physical signs of dilatation of the stomach?

Dilatation of the stomach gives rise to abnormal prominence of the upper portion of the abdomen, with extension of the area of gastric percussion-tympany when the organ is empty, and of percussion-dulness when it is filled. When partially filled (the ordinary condition), succussion-splashing is easily elicited, the stethoscope being placed over the pyloric region while sudden motion is imparted to the left hypochondrium and lumbar region. If so late as thirty-six hours after the administration of salol, salicylic acid can still be detected in the urine by a purplish or purplish-brown precipitate on the addition of tincture of ferric chloride, the delay in the propulsion of the stomach-contents into the duodenum is considered sufficient to warrant the diagnosis of gastrectasia.

Gastric Ulcer.

What are the symptoms of gastric ulcer?

Destruction of the mucous coat of the stomach may be a result of acute gastritis. More commonly, however, gastric ulceration develops in the course of chronic gastric catarrh, especially in anemic women with impaired nutrition. In some cases, ulcers of the stomach result from occlusion or obstruction of gastric vessels. Gastric ulcer is not infrequent in cases of cirrhosis of the liver. The symptoms are sometimes obscure, and the disease may go unrecognized until suddenly perforation and death result. Symptoms of gastric catarrh are usually present, with vomiting, impaired appetite, discomfort after meals, flatulence, acidity, coated tongue. There are acute pain and marked tenderness in the epigastrium or hypochondrium, also sometimes in the lower dorsal or upper lumbar region. Pain and tenderness over or near the spinal column opposite the site of epigastric pain is quite characteristic of gastric ulcer. The pain and accompanying tenderness, anteriorly as well as posteriorly, are most frequently distinctly circumscribed. Pain is aggravated by the ingestion of food, especially solid food, and relieved by vomiting, which usually occurs soon after food is taken. The vomited matters are often blood-streaked ; or a considerable quantity of blood, bright-red in hue, or discolored by the gastric juice, is vomited. Following hematemesis the stools contain more or less black, tarry matter. Anemia is a common attendant upon gastric ulcer. Dyspnea and palpitation are its concomitants. There are often great emaciation and profound prostration. Occasionally perforation of the walls of the stomach results ; if adhesive inflammation have occurred, adjacent structures, as liver or pancreas, may constitute the floor of the ulcer ; otherwise fatal peritonitis ensues.

How is chronic gastritis to be distinguished from gastric ulcer?

The difference is one of degree. With ulceration of the stomach are associated the symptoms of chronic gastritis, but in addition there are the exquisite localized epigastric pain and tenderness, which differ from the diffuse soreness of simple gastritis. Hematemesis is usual in ulcer of the stomach and less usual in chronic gastritis of other than alcoholic origin ; and the blood in the latter case is not likely to be bright in color or of frequent appearance in the vomit. In gastritis vomiting occurs not only after taking food, but not uncommonly on an empty stomach as well. The vomiting of ulceration is usually brought on by eating. Chronic gastritis responds more readily than gastric ulcer to judicious treatment. The age and sex of the patient sometimes help the diagnosis, as ulcer is most frequent in young persons and especially in anemic girls.

Carcinoma of the Stomach.

What are the symptoms of carcinoma of the stomach?

Carcinoma of the stomach appears principally in two forms. In the one the cellular element predominates ; in the other the fibrous. The former involves the coats of the body of the stomach; the latter almost exclusively the pylorus. The *symptoms* occasioned differ in each case. When the body of the stomach is involved the symptoms are those of an aggravated chronic gastritis: impaired appetite, pain after meals, sometimes vomiting, with slow emaciation. As time goes on, ulceration takes place in the neoplasm, with the addition of the symptoms of this condition: severe, deep-seated pain in the region of the epigastrium, aggravated by the ingestion of food, vomiting of blood-streaked matters and of discolored blood presenting an appearance of coffee-grounds. The stools contain tarry matters, from disorganized blood. With the infiltration of the walls of the stomach, adhesions are formed with adjacent organs, which may be progressively invaded.

When the new-growth involves the pylorus, a characteristic feature is dilatation of the stomach. The organ may be considerably displaced by the weight of the tumor. Emaciation ensues; while the development of the carcinomatous cachexia is of diagnostic significance. The feature without the establishment of which the diagnosis of carcinoma of the stomach is doubtful is the presence of a tumor in the epigastrium. In scirrhus of the pylorus death results from exhaustion, as a result of inanition; in cellular carcinoma metastasis plays a prominent part in determining a fatal issue.

How are carcinoma and ulceration of the stomach to be differentiated?

The similitude between the symptoms of ulcer of the stomach and those of carcinoma of the stomach is sometimes so great that the diagnosis is extremely difficult. The detection of a tumor in the area occupied by the stomach is convincing. In its absence, a cachexia or the detection of enlarged glands or new-growths at other parts of the body may afford corroborative evidence. While the symptoms of ulceration may set in acutely, and rapidly assume a grave aspect, they may on the other hand be comparatively mild; recovery is the rule. The symptoms of carcinoma are more slowly developed, more persistent and more profound than those of ulcer, and the progress of the case reveals its malignancy. If the pylorus is involved, vomiting does not take place for some time after food has been taken, while both in ulceration and in cellular carcinoma of the body of the stomach vomiting, when it occurs, takes place early. Obstruction of the pylorus by carcinoma occasions dilatation of the stomach. Before the fortieth year of life ulceration is the more common; after forty, carcinoma. In ulcer, the gastric hydrochloric acid is said to be excessive; in carcinoma, to be wanting. In simple ulcer, sudden, more or less profuse hemorrhages occur; hence the blood is often bright, and when discolored it is a viscid fluid or coagulated in coherent clots. In carcinomatous ulcer there is a slight but more or less continuous oozing; hence vomiting of blood rarely occurs apart from the admixture with food of the disorganized "coffee-grounds" sediment.

How is carcinoma or sarcoma of the omentum to be distinguished from carcinoma of the stomach?

The detection of a tumor in the region of the epigastrium is not exclusively indicative of carcinoma of the stomach. The omentum may be the seat of carcinoma or sarcoma. In such a case the symptoms of gastritis are not necessarily present; in particular the vomiting of matter resembling coffee-grounds is wanting.

How is carcinoma of the stomach to be distinguished from carcinoma of the pancreas?

It may be impossible to decide from anatomic considerations, especially in view of the displacement that often occurs, whether a tumor in the epigastrium is gastric or pancreatic. If the former, definite symptoms of gastric derangement are present; the visceral symptoms of the latter are ill-defined. The existence of diabetes and the imperfect digestion of fats would point to involvement of the pancreas. Jaundice sometimes accompanies carcinoma of the pancreas from pressure upon the bile-ducts. In gastric carcinoma without hepatic complication this does not occur.

How is chronic gastritis to be distinguished from carcinoma of the stomach?

It is not sufficient to make a diagnosis of chronic gastritis. The existence of carcinoma should, if possible, always be excluded. If, in addition to the symptoms of gastritis, there is severe and persistent pain in the epigastrium, or pain increased or developed after eating, with vomiting shortly after meals or after a variable interval, the ejected matters resembling coffee-grounds; if there exist cachexia, new-growths in various parts of the body, and a tumor can be detected in the region of the epigastrium, the gastritis is but a concomitant of the malignant disease, which, in the course of a year or eighteen months, is almost necessarily fatal. Carcinoma is uncommon before forty; gastritis may occur at any time of life. The gastric juice is said to contain free hydrochloric acid in nearly all cases of chronic gastritis, but to contain none in cases of gastric carcinoma. While the persistent absence of free hydrochloric acid should excite suspicion of the existence of malignant disease, it is not conclusive evidence.

THE INTESTINES.

Acute Enteritis.

What are the symptoms of acute enteritis?

Acute inflammation of the small intestine may vary greatly in severity, from a simple catarrh of the mucous lining to an intense inflammation involving the submucous and muscular tunics and even the peritoneal investment.

Acute intestinal catarrh or *mucous enteritis* is, in most cases, dependent upon the presence of irritating matters in the bowel. As a result, there occur diarrhea, with colicky pains, and often considerable tenderness in the abdomen, and febrile and other constitutional manifestations of varying intensity. The *stools* may number from three to six or more in twenty-four hours; they are thin and liquid, containing undigested food and considerable mucus, and may be streaked with blood. The stomach is likely to share in the inflammatory process, and the symptoms of a more or less intense gastritis are superadded.

When *gastro-enteritis* is due to the ingestion of corrosive and poisonous substances or to the ingestion or development of ptomaïnes, the symptoms become more severe, and collapse and death may occur.

In the severe cases of *acute enteritis*, involving the serous, muscular and submucous coats, constipation, or constipation alternating with slight, irritative diarrhea, is the rule; actual obstruction of the bowel, from inflammation, paralysis, or incarceration by bands of lymph, is not uncommon. The constitutional and local symptoms are correspondingly intense. There may be an initial chill. While the pain may be colicky at first, it soon becomes constant, subject, however, to paroxysmal exacerbations. It is increased by pressure, tenderness at times being exquisite. As in peritonitis, the patient lies upon his back with flexed thighs, to relax the abdominal muscles. There is not uncommonly a marked and distressing pulsation to the right of the umbilicus. Thirst, nausea, vomiting and retching may be decided, even in the absence of gastric involvement. The *fever* becomes high and does not remit, as it does in intestinal catarrh. The *pulse* is rapid; at first tense and full; afterward, small and wiry. Following a copious stool, amelioration and recovery may ensue; or symptoms of failing circulation, with distention of the abdomen, hiccough, incessant retching, sweating, anuria and exhaustion, may precede death.

How are acute catarrhal enteritis and typhoid fever to be differentiated?

Some degree of inflammation of the small intestine necessarily attends typhoid fever. Occurring as an independent affection, however, enteritis is wanting in the epistaxis, the severe headache, the rose-spots, the characteristic temperature-curve, the peculiar character of the stools, the gravity and the typical course and duration of the general infectious disease.

Croupous Enteritis.

What is croupous enteritis?

Croupous or *membranous enteritis* is a somewhat rare disease, characterized by the discharge of fibrinous casts of the bowel, or of flakes of false membrane.

These are usually expelled, after paroxysms of colic, with painful straining, in watery stools containing mucus, sometimes blood, but little fecal matter. The paroxysms are often preceded by constipation, and associated with or followed by diarrhea. The affection is obstinately recurrent. It occurs chiefly, if not exclusively, in hysterical or hypochondriacal subjects in early adult or middle life.

Cholera Morbus.

What are the symptoms of cholera morbus or cholera nostras?

Cholera morbus or *cholera nostras* is an acute disease, most prevalent during the summer months and characterized by inflammation of the stomach and intestines. It is commonly a result of the ingestion of unsuitable and irritating articles of diet. The affection is manifested by colicky pains in the abdomen, by nausea, vomiting and diarrhea, by cramps in the legs, by increased thirst, by headache, vertigo and debility, by coldness of the extremities, by prostration and rapid wasting. Despite the severity of the symptoms, recovery is almost invariable.

How are cholera morbus and cholera Asiatica to be differentiated?

The symptoms of cholera morbus and those of cholera Asiatica differ principally in degree. Cholera Asiatica, however, occurs in epidemics; isolated cases are rare. Cholera morbus, on the other hand, is not an epidemic disease. The mortality from cholera Asiatica is high; recovery from cholera morbus is the rule. The detection of characteristic comma bacilli in the feces or in the vomit confirms a diagnosis of cholera Asiatica.

Cholera Infantum.

What are the symptoms of cholera infantum?

Cholera infantum is practically gastro-enteritis in children. It is a disease of the summer months, and is intimately related with heat, foul air, uncleanliness and fermentation of food. It is manifested by vomiting, profuse watery, often fetid diarrhea, fever, rapid wasting, depressed fontanel, convulsions and coma. Among the poor the fatality of the disease is great.

Chronic Enteritis.

What are the symptoms of chronic enteritis?

As *chronic enteritis* or *intestinal catarrh* most frequently results from persistent errors in diet, the symptoms depend upon retarded and defective intestinal digestion. Pain and oppression occur at a time after the ingestion of food that varies with the seat of the morbid process. In duodenal catarrh the symptoms appear earlier than when the jejunum or ileum is involved. When the lower portion of the intestine is affected, colicky pains are frequent. There may be persistent diarrhea, obstinate constipation, or diarrhea alternating with constipation. The stools are slimy and may contain undigested food. The *abdomen* is distended; flatulence and belching are usual. The *complexion* is sallow. The *nutrition* is impaired. Headache is common. *Sleep* is disturbed by dreams, while there is unusual drowsiness.

Acute Dysentery.

What are the symptoms of acute dysentery?

Acute dysentery is practically an inflammation of the large intestine, probably dependent upon a specific infection; rarely the lower portion of the small intestine is involved. The inflammation may be of varying intensity. It may be catarrhal, ulcerative or diphtheritic. There are slight fever, abdominal pains of a griping character (tormina), terminating with frequent, small, slimy stools, streaked with blood, each evacuation being attended with bearing-down, burning pain and muscular spasm (tenesmus). The stools sometimes contain pus, sometimes shreds of membrane; in many cases the ameba coli is found. In addition there are headache, vertigo, weakness, thirst and perhaps nausea and vomiting. Dysentery is a prolific source of hepatic abscess. Death may result from exhaustion, or from perforation of the bowel.

How are intussusception of the bowel and acute dysentery to be differentiated?

In cases of intussusception of the bowel the stools may be small, frequent, mucous, blood-streaked, and attended with tenesmus. Inquiry, however, will elicit a history of abrupt onset; examination will disclose the presence of a sausage-shaped abdominal tumor, perhaps also protrusion of the bowel at the anus. Intussusception is the more common in children; dysentery, the more common in adults.

How are acute enteritis and acute dysentery to be differentiated?

The pain of acute intestinal catarrh is colicky, but the peculiar tormina and tenesmus of dysentery are wanting. The stools of dysentery are more frequent than those of enteritis, are smaller in quantity, contain more blood, and are in less degree fecal. Dysentery is more likely than is enteritis to be epidemic.

How are acute dysentery and typhoid fever to be differentiated?

While both acute dysentery and typhoid fever are attended with diarrhea, the stools of dysentery are frequent and small, perhaps ineffectual, and are composed principally of blood-streaked mucus; the stools of typhoid fever are not necessarily frequent, are larger, often thin and like pea-soup. The epistaxis, the rose-spots and the characteristic temperature-curve of typhoid fever are wanting in acute dysentery. The duration of dysentery is briefer than that of typhoid fever.

Chronic Dysentery.

What are the symptoms of chronic dysentery?

Dysentery may from the outset manifest a tendency to chronicity; or an acute dysentery may become chronic. The symptoms of chronic dysentery differ little from those of the acute disease. Febrile symptoms, however, are wanting; intermissions occur; wasting takes place; anemia develops; the complexion becomes sallow; hepatic abscess may form.

Typhlitis.

What are the symptoms of typhlitis?

Inflammation takes place in the cecum or vermiform appendix as a result of the accumulation and impaction of fecal matters; or from irritation by a calculus, the nucleus of which may be inspissated mucus from catarrh of the appendix; or by a foreign body, such as a cherry-stone or a grape-seed. Traumatism and straining are occasional exciting causes. The process may be catarrhal, ulcerative or gangrenous. The premonitory symptoms are vague and are frequently mistaken for simple colic. Diarrhea may alternate with constipation, still further misleading the inattentive observer. If the cecum be involved, it becomes paralyzed and distended with accumulated feces; thus, the condition declares itself primarily by the symptoms of intestinal obstruction, by pain, often severe, and increased by motion, in the right iliac fossa and right hip, with tenderness, a sense of doughy induration, and dulness on percussion. The tumor is superficial and sausage-shaped, its long axis pointing inwards and downwards. It is slightly movable; gurgling may sometimes be developed. The pain may be paroxysmal or paroxysmally aggravated, and is frequently of an agonizing character. There is usually some fever, and at times the temperature may reach from 102° to 104° F. Peritonitis may develop, even without ulceration and perforation of the bowel, and its symptoms then predominate. It may remain localized or become diffused. With relief to the obstruction, recovery may ensue, or collapse may suddenly occur, recovery or death following.

If *appendicitis* alone exists there may be no interference with the passage of the intestinal contents, and the evidences of a tumor in the iliac fossa are wanting, unless the appendix have undergone great distention. In ulcerative appendicitis, perforation not rarely results, giving rise to a general or localized purulent peritonitis. This event may be announced by sudden pain, shock, chill and rise of temperature, followed by the development of a fluctuating tumor indicative of abscess; or the condition may develop insidiously and be difficult of recognition. Sometimes, after initial shock and pain, deceptive improvement is manifested. Surgical exploration may be a diagnostic necessity. Attacks of typhlitis are prone to be repeated.

Perityphlitis.

What are the symptoms of perityphlitis?

Perityphlitis, or inflammation of the tissues surrounding the cecum and its appendix, usually occurs in the course of typhlitis or of appendicitis. The fibrous structures and the peritoneum are involved alone or in association. If perforation takes place, an abscess may form, or general peritonitis result, or both complications may be present. As a rule, the pus is shut off from the general peritoneal cavity by a capsule. The symptoms vary with the pathologic association. To those of the primary condition are added an acute exacerbation of pain and tenderness, as well as of the general symptoms, perhaps preceded by a chill. The pain is deep-seated and is increased by flexing the right thigh upon the abdomen. Sometimes the patient is unable to lift the right leg. He usually lies upon the right side, with the thigh semiflexed. If an abscess forms, there may be repeated rigors and a fluctuating tumor in the right iliac fossa. The tumor is not superficial and sausage-shaped like that of cecitis, but is deep-seated and irregular. A pericecal abscess may sometimes be detected by rectal exploration. If peritonitis develop, death may ensue, from septicemia or gradual exhaustion, or suddenly, with manifestations of collapse.

With what conditions may appendicitis or perityphlitis be confounded?

Inflammations of the cecum and its appendix or inflammation and abscess of the surrounding tissue have been mistaken for typhoid fever, and for "idiopathic peritonitis."

The mistake is more likely to be made when the inflammation of the appendix has been slow and the symptoms indistinct, until perhaps perforation occurs, causing a limited abscess or a general septic peritonitis. Deep-seated but limited abscess may for a time be concealed from other than surgical exploration, and through septic poisoning give rise to the "typhoid state."

Before localizing symptoms, such as induration, or the presence of a fluctuating tumor in the iliac fossa, render the case clear, the occurrence of one or more rigors, the absence of distinctive characteristic symptoms of typhoid fever, the course of the temperature, and sometimes the location of the tenderness, should prevent mistake. The location of the pain and tenderness, however, may be misleading, as the appendix varies greatly in its position; and appendicitis has even been known to simulate hepatic disease. Sometimes only an exploratory incision can settle the diagnosis.

The exploring needle or aspirator should never be used.

How are perityphlitis and typhlitis to be distinguished from a lumbar abscess?

Destructive disease of a lumbar vertebra is usually followed by suppuration, the pus following in the course of the psoas muscle and seeking exit below Poupart's ligament. A collection of pus forming in this way differs from typhlitis or perityphlitis by the absence of symptoms of intestinal derangement. The situation of the tumor is different in each case. In the one case, examination will reveal a deformity of the spinal column, with pain and tenderness in the lumbar region. The symptoms are slow and progressive. They may be associated with visceral tuberculosis.

How are typhlitis and perityphlitis in a woman to be distinguished from an abscess of the right ovary?

An abscess of the right ovary is situated nearer the middle line than is the swelling of typhlitis or perityphlitis. With typhlitis or perityphlitis is associated gastro-intestinal derangement; with abscess of the ovary, uterine and menstrual derangement. Vaginal and rectal examination may clear up any doubt.

How are typhlitis and perityphlitis to be distinguished from carcinoma of the cecum?

The symptoms of typhlitis and perityphlitis are likely to appear suddenly; those of carcinoma insidiously and progressively. Typhlitis and perityphlitis are affections of comparatively brief duration; carcinoma of the cecum will probably continue for a number of months after its detection. The inflammatory processes are usually attended with fever; carcinoma is not. When typhlitis or perityphlitis has existed for some time, fluctuation—indicative of the occurrence of suppuration—can be detected in the tumor; the carcinomatous new-growth retains its original density. Perityphlitis or typhlitis may be attended with emaciation and sallowness of skin, but not with the cachexia of carcinoma. When the cecum is the seat of carcinoma, like new-growths are usually found in other parts of the body.

Intestinal Obstruction.

What are the symptoms of intestinal obstruction?

The lumen of the bowel may be obliterated by an accumulation of feces, by a large gall-stone, by an intestinal calculus (enterolith) or other foreign body, by organic narrowing of the bowel, by stricture or neoplasm, by a twist or volvulus, by external constriction and by strangulated hernia, internal or external.

The condition may arise in a subject of habitual constipation or of hernia. It may follow a violent physical effort. It may be due to acute enteritis. From the onset, or after a variable period during which no stool has been passed, abdominal pain and rumbling set in. Vomiting ensues; at first of the contents of the stomach, then of yellowish-green fluid and mucus; finally the vomiting becomes stercoraceous. The apparent constipation does not submit to ordinary measures. The abdomen becomes distended. The expression of the face is drawn and anxious, the pulse small, rapid and feeble, the surface cold and clammy, and if the condition be not relieved by medicine or operative intervention, death is the inevitable result.

When the obstruction is not complete, as in some cases of fecal impaction, there may be more or less frequent passages of liquid matters somewhat fecal, which the patient will describe as diarrhea. Inspection of the stools, and the evidences given by palpation and percussion, of the presence of a hard mass in the course of the bowel, usually in the transverse or descending colon, will prevent error. The nature of the obstruction in any case is to be determined principally by physical examination. In cases of strangulated hernia the knowledge of the existence of hernia may assist the diagnosis. In the absence of such history, examination must none the less be made, if only to exclude that condition from among the possibilities in the case.

Intussusception.

What are the symptoms of invagination or intussusception of the bowel?

Under certain circumstances, not definitely recognized, one portion of the bowel becomes invaginated in another portion. The small intestine or the large intestine, respectively, may be alone involved; but most commonly the small intestine enters the large at the ileo-cecal orifice. The occurrence of the accident is announced by a sudden attack of pain, repeated in paroxysms, followed by the presence of a sausage-shaped tumor in the abdomen, and stools of a dysenteric character. Sometimes no fecal matter is passed, and there are frequent discharges of blood-stained mucus. The pain is intense, and the child (for the affection is most common in children) often draws up its legs close to the belly. Pressure and manipulation relieve the pain, and quiet the excruciating cries of agony. In the course of a variable period of time, blood is passed by the bowel, the pain becomes continuous and vomiting occurs, with the symptoms of intestinal obstruction. The invaginated bowel may be [illegible] (per rectum); it may even protrude from the anus, or may slough off and be discharged, and recovery ensue; or it may [illegible] of the bowel. Intussusception is more common in children than in adults, and in boys than in girls.

How are intussusception and obstruction of the bowel to be differentiated?

Intussusception is the more common in children; obstruction, in adults. The symptoms of intussusception appear abruptly; those of obstruction are often abrupt in onset, though sometimes of long standing. A sausage-shaped tumor is characteristic of intussusception; certain varieties of obstruction are attended with abdominal tumors of irregular shape. Constipation is not so absolute in intussusception as in obstruction; and stercoraceous vomiting is less common in the former than in the latter. A discharge of blood and the protrusion of a portion of bowel from the anus are diagnostic of intussusception. Digital exploration of the rectum will often assist in discrimination.

How are typhlitis and intussusception of the bowel to be differentiated?

Both typhlitis and intussusception may present a sausage-shaped tumor in the right iliac fossa and be attended with severe pain and the evidences of intestinal obstruction; but intussusception, unlike typhlitis, is sudden in onset, is uncommon in adults, is usually afebrile, is likely to be attended with ineffectual or bloody stools, and perhaps to be accompanied by protrusion of the bowel at the anus. The tumor of intussusception is not necessarily confined to the right iliac fossa.

Carcinoma of the Intestine.

What are the clinical manifestations of carcinoma of the intestine?

Carcinoma of the intestine is most commonly situated in the rectum, the sigmoid flexure, the cecum, the vermiform appendix or the duodenum. When in the duodenum, the papilla of the pancreatic duct and common bile-duct is usually involved and jaundice is apt to result. When other parts of the bowel are involved, in addition to the pain and constitutional phenomena occasioned by the malignant growth, symptoms of partial intestinal obstruction develop. There is obstinate constipation, and when the bowels are moved, the stools appear as thin, flat bands, often streaked with pus and blood. On physical examination a tumor may be detected.

Intestinal Parasites.

What are the most common varieties of intestinal parasites?

The most common intestinal parasites belong to the order of vermes, of which there are two important classes—*cestodes*, or tape-worms, and *nematodes*, or round-worms.

Of the former the more important are the *tenia solium*, the *tenia mediocanellata*, and the *bothriocephalus latus*.

Of round-worms the most common are the *ascaris lumbricoides*, the *oxyuris vermicularis*, and the *trichina spiralis*.

The oxyuris vermicularis inhabits the large intestine, the tape-worms and the lumbricoides the small intestine; the trichina migrates from the stomach and small intestines into the muscles, setting up an irritative fever with special symptoms, the condition being known as *trichiniasis*.

What symptoms are occasioned by the presence of animal parasites in the intestinal canal?

The presence of worms in the intestinal canal of an otherwise healthy individual may occasion no appreciable disturbance. In other cases, however, there are evidences of gastro-intestinal derangement, capricious appetite, abdominal uneasiness, colicky pains, possibly diarrhea, nausea, vomiting, loss of flesh, debility, cachexia, irregular fever, disturbed sleep, gritting of the teeth, itching of the nose and anus, nervous manifestations, even epileptiform convulsions.

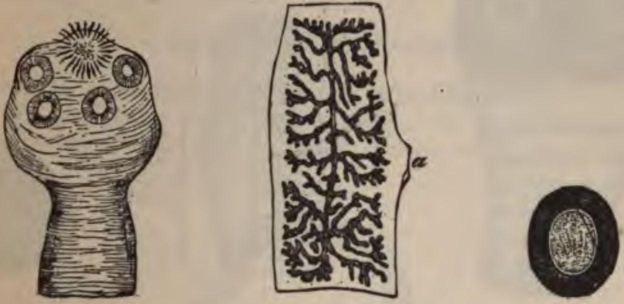
The diagnosis of intestinal worms depends upon the discovery of the parasites or of their ova in the stools.

Tenia Solium.

What are the characteristics of the tenia solium?

The *tenia solium* (Fig. 24) is a tape-worm having a small head, or scolex, and a slender neck. The head is of the size of a pinhead, and is surmounted by a circle of twenty-six hooklets, around which are four suckers. From the neck pass off segments, or proglottides, that progressively increase in size. The entire worm, or strobila, may be from seven to ten feet long and composed of from four hundred to six hundred segments. The tenia solium usually develops in man from the ingestion of "measly" pork or the flesh obtained from swine infected with the cysticercus cellulosæ, which in turn develops in animals that have swallowed the ova of the tenia solium.

Fig. 24.



Head. Mature segment. Ovum.

Tenia Solium, magnified (Heller).

Tenia Mediocanellata.

What are the characteristics of the tenia mediocanellata?

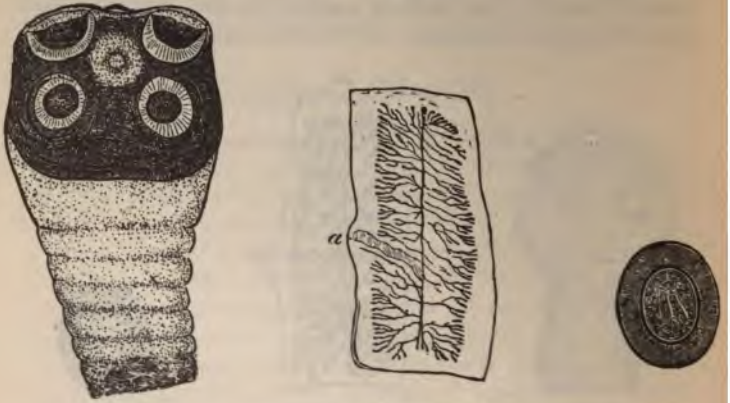
The *tenia mediocanellata*, or *unarmed tape-worm* (Fig. 25) differs from the tenia solium in that the head, though surmounted by four suckers, is without hooklets. The tenia mediocanellata may attain a length of from ten to twenty feet, and be composed of from eight hundred to more than one thousand segments, which are longer and broader than those of the tenia solium

The tenia mediocanellata is transmitted to man by the raw flesh of sheep or cows, in which hosts the larvæ develop from the ova of the tenia mediocanellata.

Tape-worms are usually present in small numbers; there is often but a single worm; sometimes there are two or three.

About three months are requisite for the development of a tape-worm.

FIG. 25.



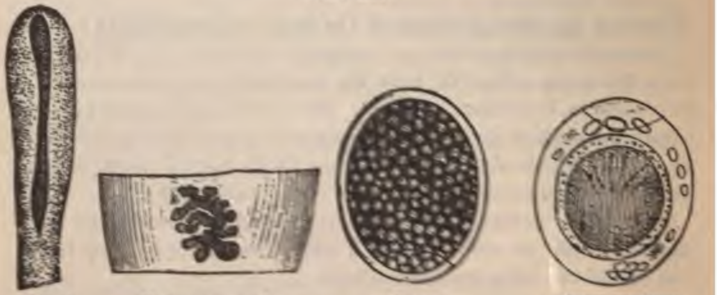
Head. Mature segment. Ovum.
Tenia mediocanellata, magnified (Heller).

Bothriocephalus Latus.

What are the characteristics of the bothriocephalus latus?

The *bothriocephalus latus* (Fig. 26) sometimes called the *tenia lata*, is a cestode worm, with a club-shaped head and a filament-

FIG. 26.



Head. Mature segment. Ovum. Ovum: embryo developed.
Bothriocephalus latus, magnified (Heller).

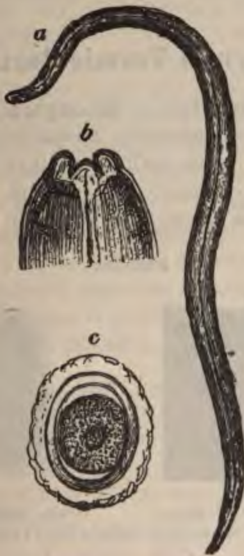
ous neck. On either side of the head is a longitudinal sucker. The mature segments present a characteristic stellate appearance, dependent upon the distention of the uterus with ova. The worm may be from fifteen to twenty-five feet in length, and constituted of from three thousand to four thousand segments. It is thought to be derived from fish or fresh-water molluscs.

Ascaris Lumbricoides.

What the characteristics of the ascaris lumbricoides?

Lumbricoid or *round worms* inhabit the small intestine. Small worms, embryos, or ova, are supposed to gain access to the alimentary canal of man through drinking water, and in some instances to be conveyed by the fingers to the mouths of

FIG. 27.



a, natural size; *b*, head, magnified; *c*, ovum, magnified.
Ascaris lumbricoides (v. Jaksch).

those engaged in cleansing privies or otherwise handling excrement, but the exact history of their development is unknown.

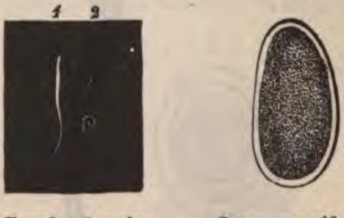
Mature worms are from eight to fifteen inches in length, are attenuated at both extremities, and resemble common earthworms. Lumbricoid worms may be present in varying numbers, from one to a dozen or more. They are usually multiple, and in rare instances may be so multitudinous as to occlude the intestine. The worms may find their way into the stomach, and be expelled by vomiting. They may pass from the esophagus into the larynx and trachea. They have in this way caused suffocation in children. They may cause occlusion of the bile-duct or pancreatic duct, and, though rarely, suppuration of the liver or of the pancreas. They may also leave the intestine by way of a perforation, causing peritonitis or fecal abscess.

Oxyuris Vermicularis.

What are the characteristics of the oxyuris vermicularis?

Oxyures vermiculares, *seat-worms*, *thread-worms*, or *spool-worms*, are from one-eighth to one-half of an inch long. They infest the large intestine, especially the rectum. The worms find their way out of the anus, and give rise to intense itching. Sometimes they gain access to the vagina, and occasion unpleasant symptoms. The parasites may be present in the bowel in large numbers; they are often found in the stools in tangled masses, resembling bunches of thread.

FIG. 28.



1, Female; 2, males. Ovum, magnified.
Oxyuris vermicularis, natural size (Vierordt).

Acute Peritonitis.

What are the symptoms of acute peritonitis?

Acute peritonitis may result from the entrance of noxious matters into the peritoneal cavity, either through the blood, by extension from adjacent disease, by intestinal perforation, or by injury of the abdominal walls. Thus, among its causes may be chronic nephritis, acute rheumatism, typhoid fever, dysentery, typhlitis, appendicitis, disease of the uterus and oviducts, tuberculosis, infectious diseases and traumatism.

Acute peritonitis may be *local* or *general;* it may be *simple*, *purulent*, or *putrid*. It is attended with acute abdominal pain, tympanites, nausea, vomiting, constipation, considerable elevation of temperature and other febrile manifestations. The *pulse* is small, rapid and tense—"wiry." The *face* is drawn; the *expression* anxious.

The *breathing* is shallow, rapid, and thoracic. The abdominal pain and tenderness are so intense that the patient shrinks from the slightest movement, and complains of the lightest covering. The legs and thighs are drawn up in flexion, to relax the abdominal parietes. The surface of the body may be covered with a cold sweat and collapse may ensue. Effusion may take place or adhesions form among the structures in the peritoneal cavity. Should pus form, there are repeated rigors and hectic fever. If the inflammation is putrid, death speedily takes place amid the symptoms of profound intoxication.

How are acute gastritis and acute peritonitis to be distinguished from one another?

Nausea, vomiting, constipation, headache, abdominal pain and tenderness, and febrile symptoms attend both acute gastritis and acute peritonitis. In the former, however, the vomiting occurs earlier and is more aggravated than in the latter, and the vomited matter may contain blood. In peritonitis, the abdominal pain and tenderness are not confined to the epigastrium, but are more extensive and more intense than in gastritis, while abdominal distention is more decided. A cause will be obvious for an acute gastritis sufficiently intense to simulate peritonitis, the symptoms of which are relatively the more profound.

How are acute peritonitis and acute enteritis to be differentiated?

The pain of enteritis is colicky; that of peritonitis, lancinating. Tenderness is greater and more general in peritonitis than in enteritis. Diarrhea is common in enteritis; constipation is the rule in peritonitis. Nausea and vomiting are more decided in peritonitis than in enteritis. Effusion occurs in peritonitis, not in enteritis. The constitutional disturbance of peritonitis is comparatively more profound than that of enteritis. Rigors and fluctuating temperature are suggestive of peritonitis. The cause of peritonitis is sometimes obvious in the history of the case, or discoverable upon vaginal or other examination.

How are acute intestinal obstruction and acute peritonitis to be differentiated?

Constipation attends both acute peritonitis and acute intestinal obstruction; it may yield in the one, but it is insuperable in the other. Prior to the acute symptoms of obstruction there may have been small, liquid evacuations, but, when the symptoms of obstruction have set in, constipation is absolute. The vomiting of acute peritonitis does not present any unusual features; that of intestinal obstruction soon becomes stercoraceous. The pain of obstruction is colicky; that of peritonitis is sharp and lancinating. The exquisite abdominal tenderness of peritonitis is not encountered in obstruction. When obstruction exists, the rolling of the intestines may be apparent to the eye or to the palpating hand. Peritonitis occasions paralysis of the bowel. The febrile symptoms of peritonitis are wanting in uncomplicated obstruction.

Inquiry and physical examination may elicit one of the known causes of peritonitis or of obstruction respectively.

How are acute peritonitis and intestinal colic to be differentiated?

Acute peritonitis and intestinal colic have pain in common; the latter, however, is unattended with febrile manifestations.

The pain of colic is inconstant and is relieved by frictions; in peritonitis the pain persists and is intensified by the slightest touch.

Colic, as a rule, is symptomatic, and its cause is to be ascertained upon careful search. A blue line on the gums would indicate lead-poisoning.

How is subacute rheumatism involving the muscles of the wall of the abdomen to be distinguished from acute peritonitis?

The pain of rheumatism is not so severe as that of peritonitis; nor is it as constant; nor does it give rise to symptoms of gastro-intestinal derangement; and it is not attended with febrile manifestations; while in addition there are other evidences and a history of exposure to influences productive of subacute rheumatism.

Chronic Peritonitis.

What are the symptoms of chronic peritonitis?

Chronic inflammation of the peritoneum may be the sequel of an acute attack; it may be insidious in development. It is commonly a result of persistent irritation, such as may depend upon the presence of new-growths, carcinomatous, sarcomatous or tuberculous, or upon chronic inflammation of the abdominal or pelvic viscera.

Adhesions form between adjacent viscera and fluid collects in the abdominal cavity. Paroxysmal attacks of pain occur. The abdomen is distended. There may be nausea and vomiting.

The action of the intestines is interfered with and constipation usually results. In tuberculous cases, with concomitant ulceration of the intestine, there may be intercurrent or continuous diarrhea. In proportion to the chronicity of the attack the omentum is found indurated and rolled up close to its attachment to the stomach, the mesentery is shortened and the lumen of the bowel is narrowed, producing visible and palpable distortions and prominences.

There is commonly more or less fever, sometimes hectic, especially if the effusion be purulent. Sometimes, particularly in tuberculous disease, there are recurrent febrile exacerbations. In any case there occur gradual emaciation and loss of strength, breathlessness, edema of the lower extremities; spontaneous recovery sometimes happens, but as a rule, unless relief be given, surgically or medicinally, death ensues. The cases are often protracted.

Malignant disease and tuberculosis of the peritoneum are most frequently associated with similar conditions in other structures.

How are chronic peritonitis and malignant disease of the liver to be differentiated?

Malignant disease of the liver, like chronic peritonitis, may give rise to a tumor in the upper portion of the abdomen, and to ascites; but the physical signs of enlargement fuse with those that normally belong to the liver; while the percussion-dulness dependent upon an omentum rolled up and contracted by chronic disease is separated from the hepatic dulness by an area ot tympanitic resonance.

Tabes Mesenterica.

What are the symptoms of tabes mesenterica?

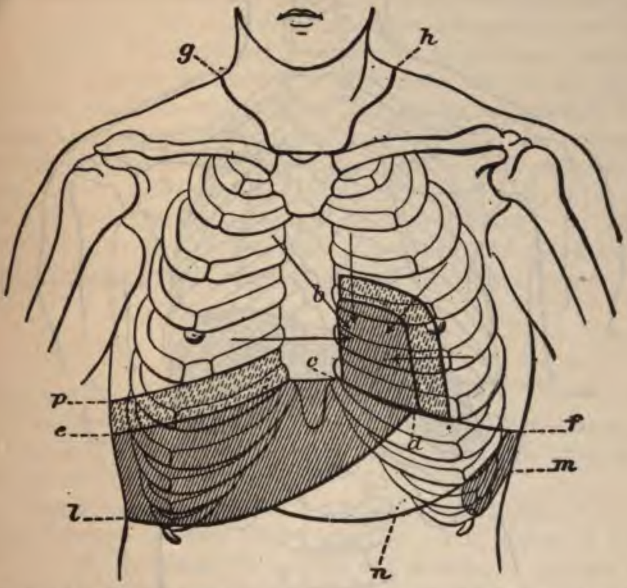
In predisposed children the glands of the mesentery sometimes become tuberculous, and there occur derangement of health, anemia, and wasting. On palpation it may be possible to detect the enlarged glands. In other respects, the symptoms are like those of subacute peritonitis and chronic enteritis. There is much confusion and dispute concerning the existence of a non-tuberculous tabes mesenterica, of which the principal objective symptom is the tumid abdomen.

THE LIVER.

What are the normal limits of the liver as determined by physical examination?

Under normal conditions the area of hepatic percussion-dulness (Figs. 29 and 30) is included between the sixth rib on the right, in the nipple line, the lower margin of the sixth rib on the right in the axilla, and the tenth rib posteriorly on the right, on the one hand, and the inferior border of the right

FIG. 29.



The relations of the heart, lungs, liver, stomach and spleen, as seen from the front (Weil). The deeply-shaded areas represent the portions of the heart, liver and spleen not covered by the lungs; the lightly-shaded areas represent portions covered by the lungs. *b*, *c*, *d*, boundary between lung and heart; *e f*, lower boundaries of lungs; *g h*, upper boundaries of lungs; *l*, lower limit of hepatic dulness; *m*, area of splenic dulness; *n*, greater curvature of stomach; *p*, upper limit of deep hepatic dulness.

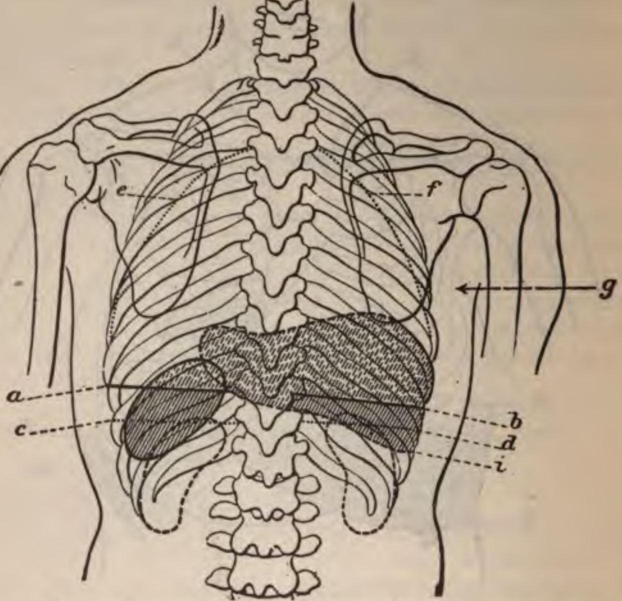
costal arch on the other. The left lobe extends into the left hypochondrium and the dulness to which it gives rise is practically inseparable from the cardiac percussion-dulness.

Floating Liver.

To what symptoms does a floating liver give rise?

Occasionally the coronary ligament of the liver becomes lengthened and the organ acquires an abnormal freedom of movement.

FIG. 30.



Showing the relations of the lungs, liver, spleen and kidneys, as seen from behind (Weil-Luschka). The deeply-shaded areas represent portions of the liver and spleen not covered by the lungs; the lightly-shaded areas represent portions covered by the lungs. *a b*, lower border of the lungs; *c d*, boundaries of complementary pleural spaces; *e f g*, divisions between lobes of lungs; *i*, lower margin of liver.

That such an unusual tumor in the abdomen is the liver is determined by the size and conformation of the organ, by the percussion-resonance in the normal region of the liver and by the absence of the phenomena of malignant disease.

Congestion of the Liver.

What are the symptoms of congestion of the liver?

Congestion of the liver results from derangement of the circulation, dependent upon disease of the heart or lungs, or upon compression of the hepatic vessels; it may result from constant irritation of the hepatic cells by improper, and especially stimulating, articles of diet; it is wont to occur in those of inactive and sedentary habits. The condition is manifested by a sense of weight and dull pain in the right hypochondrium, pain at the right shoulder, headache and vertigo. The *appetite* is impaired; the *tongue* is coated; there are nausea and vomiting; the digestion is deranged; the *bowels* are constipated; the *skin* and conjunctiva may present a yellowish tinge; hemorrhoids frequently develop; there is, sometimes, mental depression or irritability of temper; the *heart* may be irritable, as manifested by palpitation. The *urine* may contain bile-pigment and an excess of urates.

Acute Hepatitis.

What are the symptoms of acute hepatitis?

Acute hepatitis is essentially a disease of tropical climates. It may also occur in association with dysentery and other infectious diseases.

It is manifested by pain in the right hypochondrium, febrile symptoms and possibly slight jaundice. The *appetite* is impaired, and there may be nausea and vomiting. Abscess is a not uncommon sequel of inflammation of the liver.

How is the malarial cachexia to be distinguished from acute hepatitis?

As a sequel of chronic malarial poisoning, the liver and the spleen become crowded with pigment and undergo enlargement. There may be slight intermittent fever; the fever of hepatitis is continuous and not periodic. In the malarial cachexia the complexion is sallow, not jaundiced, as it may be in hepatitis. In the malarial cachexia, too, plasmodia are to be found in the blood.

How are acute hepatitis and portal phlebitis to be differentiated?

Portal phlebitis may develop in the course of infectious diseases or of pyemia; it may also result by extension from adjacent disease. It is attended by pain in the right hypochondrium, by enlargement of the liver, by distention of the veins of the abdominal wall, by ascites, by enlargement of the spleen and by diarrhea. Gastric or intestinal hemorrhage may occur. When suppuration takes place, there are recurring chills and fever, with wasting and debility. Enlargement of the spleen, distention of the abdominal veins and gastric and intestinal hemorrhage are not a part of acute hepatitis.

Acute Yellow Atrophy of the Liver.

What are the symptoms of acute yellow atrophy of the liver?

The etiology of *acute yellow atrophy of the liver* is obscure. The disease is more common in females than in males, and in young adults than in others; it sometimes appears in the course of pregnancy; at other times, in conjunction with profound emotion. After death, the liver is found to be much reduced in size and weight; in places it presents areas of reddish, purplish and yellowish discoloration, in the midst of which the hepatic cells are replaced by granules and oil-globules. By some the condition is said to be a parenchymatous inflammation of the liver.

The onset of the more grave symptoms may, for a few days or weeks, have been preceded by jaundice. This has even been associated in rare instances with enlargement of the area of hepatic percussion-flatness. Commonly, there are soon added headache, intolerance of light, delirium, stupor, convulsions and coma. The tongue is dry and coated; there are nausea and vomiting. Hemorrhages from the mucous surfaces often occur, and subcutaneous petechiæ appear. The area of hepatic percussion-dulness progressively diminishes; the abdomen becomes distended. The urine may be scarcely discolored; it contains leucin and tyrosin, and sometimes albumin; it is deficient in urea, uric acid, chlorides, phosphates and sulphates. There may be chills and irregular fever, sometimes high; but usually there is but slight elevation of temperature; and in most cases the temperature is at some time subnormal. The disease rarely lasts longer than a week; it usually terminates fatally.

How are acute yellow atrophy of the liver and typhoid fever to be differentiated?

Typhoid fever is a disease lasting three or four weeks; recovery is common. The active stage of acute yellow atrophy rarely lasts longer than a week; the termination is usually fatal. There is but slight or very irregular fever in acute yellow atrophy and nearly always a tendency to subnormal temperature; the course of typhoid fever is decidedly febrile and typical. Diarrhea is common in typhoid fever; constipation, in acute yellow atrophy. The size of the liver becomes much reduced in acute atrophy; it is unchanged or increased in typhoid fever. Rose-spots are wanting in acute atrophy, in which there may be petechiæ. Jaundice is invariable in acute atrophy; rare in typhoid fever. The cerebral symptoms are more decided in the hepatic disease than in the infective fever. The presence of leucin and tyrosin and the deficiency of urea, uric acid, chlorides, phosphates and sulphates in the urine are characteristic of acute yellow atrophy.

How are acute yellow atrophy of the liver and phosphorus-poisoning to be differentiated?

The symptoms occasioned by poisoning with phosphorus closely resemble those of acute yellow atrophy of the liver. Phosphorus-poisoning, however, usually sets in with vomiting and purging, to which the subsequent manifestations succeed; there is sometimes necrotic disease of the inferior maxillary bone. The first symptom of acute yellow atrophy of the liver is jaundice. In phosphorus-poisoning the liver is enlarged prior to becoming smaller; the diminution in the size of the liver in acute yellow atrophy is progressive from the outset of active symptoms, though the organ may have been enlarged previously. Colicky abdominal pains usually attend phosphorus-poisoning, of which it may be possible to elicit a history. The anatomic lesion of acute yellow atrophy is a parenchymatous hepatitis; of phosphorus-poisoning, a fatty degeneration of the liver.

Abscess of the Liver.

What are the symptoms of abscess of the liver?

Abscess of the liver may follow acute hepatitis; it may result from the activity of pyogenic organisms introduced into the structure of the organ in the course of ulcerative processes in the distribution of the tributaries of the portal vein or as a part of a general pyemia. When the suppuration is dependent upon hepatitis or ulceration, but a single abscess usually develops; in cases of pyemia, there may be multiple abscesses, not only in the liver, but also elsewhere. Dysentery is a prolific cause of hepatic abscess. When the two are associated, the ameba coli may be found both in the stools and in the abscess or its walls. As a result of the inflammation associated with the development of an abscess, the liver becomes enlarged and tender, the enlargement being detectable by palpation and percussion. There is pain over the liver and at the right shoulder. The right hypochondrium may be exquisitely tender. Repeated rigors may occur; there may be periodic exacerbations of temperature, followed by copious sweats. Slight jaundice may develop, but it is often absent. There may be nausea, vomiting and diarrhea. Hiccough, cough and dyspnea may result from pressure upon the diaphragm, and pleurisy, with the symptoms and physical signs denoting a rapid effusion, may result from extension of the inflammation or conveyance of the infection. If the abscess is superficial, a fluctuating tumor may be detectable in the right hypochondrium. The abscess may rupture into the pleural cavity or into the peritoneum, with a fatal termination; or the pus may make its exit through the abdominal wall, or even into the intestines. An hepatic abscess may be evacuated through the bronchial tubes. Death may result from septic poisoning or from exhaustion.

How are abscess of the liver and occlusion of the biliary passages to be differentiated?

If the common bile-duct is obstructed, bile accumulates in the gall-bladder and in the radicles of the hepatic ducts, the liver becomes enlarged, and jaundice, rigors, fever and sweats appear. The stools are pale.

If the cystic duct is occluded, there is no jaundice and the stools are not discolored. In both instances, the gall-bladder becomes distended with fluid and constitutes a fluctuating tumor, which may simulate an abscess. The differentiation depends upon the fact of the tumor occupying the situation of the gall-bladder, upon a knowledge of the existence of a cause of biliary obstruction, such as a calculus or a neoplasm, and upon the absence of a cause of abscess; nor is an abscess likely to occasion jaundice.

How are abscess of the liver and carcinoma of the liver to be differentiated?

Medullary carcinoma may give rise to a sort of fluctuation on palpation, but beyond this and the enlargement of the liver, the symptoms differ radically from those of abscess. In cases of carcinoma, chills, fever and sweats do not occur; but there is distinct cachexia, perhaps with nodules in other situations.

How is actinomycosis of the liver to be distinguished from abscess of the liver?

Actinomycosis is dependent upon the presence of a special fungus, which usually gains entrance through an abrasion of the surface, and may be disseminated by the blood-stream.

The irritation caused by the presence of the fungus, or perhaps by some product of its metabolism, is followed by inflammation and suppuration, so that the conditions of abscess are reproduced, and in that sense there is no discrimination to be made.

The diagnosis depends upon a knowledge of infection or of the existence of the disease at the original site of entrance, or the discovery of the ray-fungus (actinomyces) in the pus.

Interstitial Hepatitis—Cirrhosis of the Liver.

What are the symptoms of interstitial hepatitis, also called sclerosis or cirrhosis of the liver?

When irritating matters are in constant circulation in the blood-current, especially in the portal stream, the interstitial connective-tissue of the liver slowly undergoes hyperplasia, to the consequent detriment of the parenchymatous structures.

The rapidity of the process depends upon the intensity of the irritation. When the increase of connective tissue has reached its height, contraction sets in and the liver-cells suffer still more. The organ, at first irregularly, perhaps nodularly, enlarged, now becomes diminished in size. This is the condition of cirrhosis. The different stages are sometimes respectively called *hypertrophic cirrhosis* and *atrophic cirrhosis*. In many cases the enlargement persists, notwithstanding extensive destruction of the secreting structures. In other cases the hepatic percussion-dulness and palpation-area are apparently diminished from the first. In the early stages of the disease the symptoms are inconspicuous; there may be manifestations of derangement of the gastro-intestinal system. As the contraction becomes more marked, however, there appear evidences of interference with the functions of the liver. Ascites develops; the superficial veins of the abdomen become enlarged and prominent; this may be especially marked in the neighborhood of the umbilicus, giving rise to the so-called *Caput Medusæ;* small networks of venules appear at various parts of the surface of the body; hemorrhoids form; hemorrhages may take place from the nose and stomach; the skin assumes a pallid, clayey hue; and ultimately jaundice appears. Vertigo is not rare. Late in the disease, drowsiness and coma may develop. Excessive indulgence in alcohol is the most prolific cause of cirrhosis of the liver.

Fatty Liver.

What are the symptoms of fatty liver?

The liver may undergo *fatty infiltration* or *fatty metamorphosis.* A temporary accumulation of fat in the liver occurs under normal conditions after the ingestion of food rich in hydrocarbons. When, from any cause, oxidation becomes deficient, this fatty infiltration may become permanent and excessive, the more especially if, at the same time, immoderate eating or drinking is indulged in. Thus the condition is found in the indolent, in gourmands, in the obese, in the subjects of pulmonary tuberculosis and in drunkards.

Drunkards, however, are more liable to true *fatty metamor-*

phosis, the liver-cells in circumscribed or extensive areas undergoing oily degeneration, a condition that may likewise occur in wasting diseases, or as an apparent result of protracted discharges, or of blood-changes, in pyemia, infectious diseases, or other morbid states accompanied by protracted high temperature. It is sometimes a part of the degenerative processes of old age. It may accompany carcinoma, cirrhosis and amyloid degeneration of the liver.

The symptoms are not early obtrusive. The area of hepatic percussion-dulness is increased, and to palpation the organ seems smooth and rounded, perhaps soft and doughy. The skin may feel greasy or velvety, and is sometimes smooth and glistening; it may be pale or flushed. Jaundice is uncommon; there is no pain; ascites is rare. Diarrhea is the most constant symptom, and the least indiscretion in diet may provoke gastro-intestinal catarrh. In the more advanced stages anemia, hydremia, dyspnea and patent signs of failure of hepatic function appear.

Amyloid Disease of the Liver.

What are the symptoms of amyloid, waxy or lardaceous disease of the liver?

When the liver undergoes amyloid degeneration, other organs, such as the spleen, the kidneys, perhaps, also, the stomach and the intestines, are likewise involved, either simultaneously or consecutively. The liver is smoothly and often enormously enlarged; digestion is impaired; there may be persistent diarrhea. Pain, jaundice and ascites are uncommon. Isolated disease of the liver usually escapes detection. The process is chronic. Amyloid degeneration is a sequel of syphilis, of tuberculosis, of suppuration, of bone-disease.

How are amyloid disease and cirrhosis of the liver to be differentiated?

The liver is enlarged in both amyloid disease and cirrhosis of the liver; the enlargement is persistent in the one, replaced by contraction in the other. The amyloid liver is smooth, the cirrhotic liver usually irregular. Cirrhosis is attended with ascites and jaundice, while amyloid disease, as a rule, is not. In amyloid disease, there is a history of syphilis, of tuberculosis, of suppuration or of bone-disease; in cirrhosis, there is in most cases a history of alcoholism. With amyloid disease of the liver is usually associated amyloid disease of the kidney, occasioning the presence of albumin and tube-casts in the urine. The tube-casts will sometimes give a characteristic reaction with iodine.

Carcinoma of the Liver.

What are the symptoms of carcinoma of the liver?

The liver is a frequent seat of carcinoma, which may be primary or secondary. Primary carcinoma of the liver is usually diffuse, the organ becoming greatly increased in size; secondary carcinoma is usually nodular. In its incipiency carcinoma of the liver may escape detection. Soon, however, there is unaccountable loss of flesh, with the development of the characteristic cachexia. The liver is noted to be enlarged, and on palpation adventitious nodules may be felt through the abdominal wall, in the region of the right hypochondrium. Jaundice is uncommon, unless the biliary passages are compressed. The abdomen becomes distended and fluid collects in the peritoneal cavity. The digestive derangement becomes marked. Emaciation progresses and the patient becomes reduced to the lowest degree. There is often excruciating, lancinating pain. Tenderness in the right hypochondrium is a fairly constant attendant of carcinoma of the liver.

How is carcinoma of the liver to be distinguished from amyloid disease of the liver?

Emaciation and anemia attend both carcinoma and amyloid disease of the liver; but the straw-colored appearance presented by a patient with the carcinomatous cachexia is wanting in the conditions that give rise to amyloid disease. Cases of carcinoma do not present the peculiar distribution of amyloid disease in liver, spleen, kidneys and gastro-intestinal tract. The liver is often nodulated when carcinomatous; an amyloid liver is always smooth. Pain is present in carcinoma, absent in amyloid disease. A history of congenital, or of acquired syphilis, of tuberculoiss, or the presence of a suppurating focus anywhere in the body would weigh in favor of amyloid disease. Amyloid disease itself is not immediately fatal. Life is not likely to be prolonged for more than a year after carcinoma of the liver has been discovered.

How are carcinoma of the omentum and carcinoma of the liver to be differentiated.

When carcinoma develops in the omentum, this structure becomes shortened and rolled up beneath its attachments. As a consequence, there is dulness on percussion, and evidences of a new-growth on palpation, not alone in the right hypochondrium, but in the epigastric and umbilical regions, and in the left hypochondrium.

Carcinoma of the omentum is more commonly than carcinoma of the liver associated with ascites. In carcinoma of the omentum the symptoms of derangement of the functions of the liver are wanting. A new-growth of the liver rises and falls with this organ in expiration and inspiration; when the omentum is involved the mass is fixed.

How are carcinoma of the stomach and carcinoma of the liver to be differentiated?

Carcinoma of the stomach and carcinoma of the liver present many symptoms in common: a tumor in the right hypochondrium, progressive emaciation, cachexia and secondary growths; but when the stomach alone is involved there is obstinate vomiting, with absence of evidence of hepatic derangement; while if the liver only is involved ascites is common, and vomiting of coffee-ground material is lacking. A new-growth of the liver will rise and fall with this organ in respiration; a gastric tumor is more fixed. The percussion-dulness yielded by a tumor of the liver merges with the dulness of the liver; while the dulness of a tumor of the stomach may be separated from the hepatic dulness by an interval of tympanitic resonance. Hepatic and gastric carcinoma not infrequently coëxist.

Hydatid Cyst of the Liver.

To what symptoms does an hydatid cyst of the liver give rise?

A small, deeply-seated hydatid cyst of the liver, not interfering with the function of other parts, may give no sign. When the liver is the seat of an hydatid cyst in an accessible situation, the condition may be recognized by the presence of a soft, elastic, resistant, fluctuating tumor, the viscid contents of which may, on percussion and palpation, transmit a peculiar thrill or fremitus. This "purring tremor," recalling the trembling of a bowl of jelly, is present in about one-half of the cases. In addition, there may be pain in the right hypochondrium. The presence of such a new-growth, together with the absence of symptoms of profound constitutional disturbance, distinguishes an hydatid cyst from a malignant growth in the liver and its neighborhood.

Pressure-signs vary with the location of the tumor. A cyst situated near the hepatic duct or common bile-duct may cause obstruction and fatal jaundice; pressure on the portal vein may cause ascites; jaundice and dropsy, however, are not usual. If on the upper surface of the right lobe, the cyst will push up the diaphragm, giving rise to dyspnea and cough. The heart may be displaced. If the enlargement takes a different direction, the abdominal viscera may be encroached upon. There may be multiple cysts.

Retrogressive changes may take place in an hydatid cyst, attended with a diminution in size. The contents of the cyst may be evacuated through the stomach, bowel, bronchial tubes or abdominal wall, or into the abdominal or pleural cavity.

Spontaneous evacuation may lead to recovery or it may cause inflammation and suppuration of the invaded organ and death result. Sudden death may happen from invasion of the vena cava or of the pericardium. When retrogression or evacuation is not brought about, spontaneously or therapeutically, gradual failure and death may occur from exhaustion, or from septicemia.

How are abscess of the liver and hydatid cyst of the liver to be differentiated?

An hydatid cyst of the liver is ordinarily wanting in the constitutional phenomena of hepatic abscess: rigors, fever, sweats, emaciation. Should suppuration take place an abscess virtually results. Hydatid cysts are insidious in onset, slow in development, protracted in duration, and may undergo spontaneous disappearance; when accessible they occasion extensive percussion-dulness and a peculiar thrill on palpation. An hepatic abscess may be insidious in onset, but it soon gives rise to decided symptoms; spontaneous resolution never occurs, and thrill or fremitus is wanting. The detection of echinococcus hooklets in the contents of an hydatid cyst, and of amebæ coli in the pus from an hepatic abscess may in each case be regarded as diagnostic.

There is a growing tendency to regard exploratory puncture as inconclusive and dangerous, and to substitute exploratory incision—the latter to be followed if necessary by immediate operation.

How are an hydatid cyst of the liver and a distended gall-bladder to be differentiated?

Distention of the gall-bladder is usually dependent upon occlusion of the common bile-duct, or of the cystic duct, and is commonly associated with intense jaundice and clay-colored stools. Jaundice is exceptional in the case of an hydatid cyst, and the action of the bowels is not necessarily deranged. A distended gall-bladder occupies a definite situation; an hydatid cyst may be seated in any part of the liver. The "purring tremor" is not given by a distended gall-blader.

Perihepatitis.

What are the clinical features of chronic inflammation of the capsule of the liver, or perihepatitis?

The capsule of the liver may become thickened as a result of contiguous inflammatory processes, such as pleuritis and hepatitis, or as may be occasioned by a gastric ulcer. Perihepatitis may also be but a part of a chronic peritonitis. The liver itself is deformed, but otherwise not altered in size. The symptoms are ill-defined. Pain and tenderness in the region of the liver are usually present. Ascites develops. The kidneys are said usually to be diseased, so that the urine contains albumin.

How are chronic perihepatitis and cirrhosis of the liver to be differentiated?

Cirrhosis of the liver and perihepatitis may be associated. Occurring independently, the enlargement or the diminution in the size of the liver and the jaundice of cirrhosis are likely to be wanting in perihepatitis; while the presence of albumin in the urine points to the existence of perihepatitis.

Cholangitis—Cholecystitis.

What are the symptoms of inflammation of the bile-ducts and gall-bladder?

Inflammation or catarrh of the bile-ducts and gall-bladder, the most common cause of jaundice, is a result of the extension of inflammation or catarrh or of the invasion of microbes from the duodenum or of the presence of biliary calculi.

In consequence of the swelling of the mucous membrane, the flow of bile is interfered with and jaundice results. The surface of the body and the visible mucous membranes assume a yellowish or greenish hue; the urine is similarly discolored; the stools become scanty and clay-colored. The saliva may be discolored.

There is pain in the right hypochondrium; the liver is enlarged. The action of the heart is retarded. There is itching of the skin. The appetite is usually impaired; the tongue is coated; digestion is enfeebled; the bowels are constipated. Chills and irregular fever or transient rise of temperature may occur.

How is catarrhal jaundice to be distinguished from other forms of jaundice?

Many conditions occasion jaundice. In some, as in catarrhal jaundice, there is an interference with the discharge of bile into the intestine; in others, no such obstruction can be detected. In the first group of cases belong such conditions as occlusion of the common bile-duct by gall-stones, compression of the common bile-duct by enlarged glands or by neoplasms or as a result of inflammation and thrombosis of the portal vein. In the second group belong yellow fever, the intense forms of malarial fever, acute yellow atrophy of the liver and cirrhosis of the liver.

When the common bile-duct is occluded by a *calculus*, repeated attacks of colic are apt to occur, attended with excruciating pain, referred to the right hypochondrium; and occasionally one or more calculi are expelled into and from the intestine, so as to be found in the stools. If the calculus is large, or if more than one is present, it or they may be detected by palpation. Colic and severe pain are wanting in catarrhal jaundice, the icterus of which is apt to be less intense and less protracted than that of obstructive disease.

In *compression* of the common bile-duct, the portal vein is likely also to be compressed, so that ascites develops. When such compression is dependent upon an *enlarged gland* this may be palpable from without.

Pylephlebitis is likely to be associated with an ulcerative process in the stomach or bowel, or with a suppurating hydatid cyst and may be attended with rigors and decided febrile manifestations.

Malignant disease may involve the biliary passages or the surrounding structures. A malignant growth occasions emaciation and a peculiar cachexia manifested by a straw-colored complexion. Ascites, tumor, repeated rigors, decided fever, emaciation and cachexia are wanting in catarrhal jaundice.

Yellow fever is an epidemic, infectious disease, characterized by a train of grave symptoms, including headache, pains, fever, vomiting and prostration, that is wanting in catarrhal jaundice.

When *malarial fever* is attended with jaundice, there are commonly, in addition, chills, fever, sweats and perhaps hematuria.

The symptoms of *acute yellow atrophy of the liver* may supervene upon those of an apparently catarrhal jaundice. In such a case the area of hepatic percussion-dulness rapidly becomes diminished, cerebral symptoms appear, the urine contains leucin and tyrosin, and is deficient in urea, uric acid, chlorides, phosphates and sulphates, and death is the usual issue.

When jaundice attends *cirrhosis of the liver*, the discoloration is gradual in onset and slight in degree. There are, besides, enlargement or shrinkage of the liver and ascites.

Biliary Calculi.

What are the clinical manifestations of biliary calculi?

Biliary calculi are more common in women than in men, and late in life than early. They result from the precipitation and agglomeration of the less soluble elements of the bile. They may be single or multiple. They occasion symptoms when they give rise to obstruction.

The passage of a gall-stone through the common bile-duct is attended with excruciating colicky pain in the right hypochondrium, with shivering, nausea, vomiting and hiccough. The face is pale and the surface of the body is covered with a cold sweat. Jaundice soon develops — transient, if the calculus passes into the bowel; persistent, if the calculus remains impacted in the common duct.

By ulceration and perforation an impacted gall-stone may find its way into the stomach, the bowel or the peritoneal cavity, or it may be evacuated externally through the walls of the abdomen.

Attacks of hepatic colic are prone to be repeated. Occlusion of the common or of the hepatic duct is followed by stagnation of bile, increased secretion of mucus, and ultimately by the formation of pus. The gall-bladder may become distended into a great sac, and the accumulation advance into the biliary radicles. Under such circumstances, jaundice persists and a type of fever develops that has been designated hepatic intermittent. Periodic elevations of temperature occur, preceded by rigors and followed by sweats. Unless relief is afforded, death ultimately results from cholemia or from acholia.

Biliary calculi and carcinoma of the biliary passages are not rarely associated.

How are hepatic intermittent fever and malarial intermittent fever to be differentiated?

The intermittent fever that occurs as a result of obstruction of the biliary passages, with subsequent suppuration, closely resembles malarial intermittent fever; but in the former, there is jaundice, intense and persistent; the distended gall-bladder or the obstructing calculi may be detectable by palpation; parasites are not present in the blood; and the fever does not yield to quinine.

How are hepatic colic, renal colic and intestinal colic to be differentiated?

The pain of hepatic colic is referred especially to the right hypochondrium, whence it may radiate to the right shoulder; that of renal colic is referred to one loin, not necessarily the right, whence it radiates in the course of the corresponding ureter; the pain of intestinal colic is especially umbilical, whence it may radiate over the entire abdomen. Hepatic colic is soon followed by jaundice; the urine contains biliary pigment, and is greenish or brownish in color. In cases of renal colic, the urine may contain pus-corpuscles, blood-corpuscles and crystalline matters; jaundice is wanting. Intestinal colic depends upon intestinal derangement, sometimes upon lead-poisoning; the urine remains unaltered; there is no jaundice.

How are carcinoma of the biliary passages and obstruction of the bile-duct by calculi to be differentiated?

Obstruction of the biliary passages either by gall-stones or by carcinoma occasions persistent and intense jaundice; but in case of malignant disease there is also progressive emaciation, death usually occurring within a year of the discovery of the existence of the disease. Attacks of hepatic colic attend the presence of gall-stones in the biliary passages.

The obstruction dependent upon calculi may be overcome, spontaneously or therapeutically. Unrelieved, death ultimately results from acholia or cholemia.

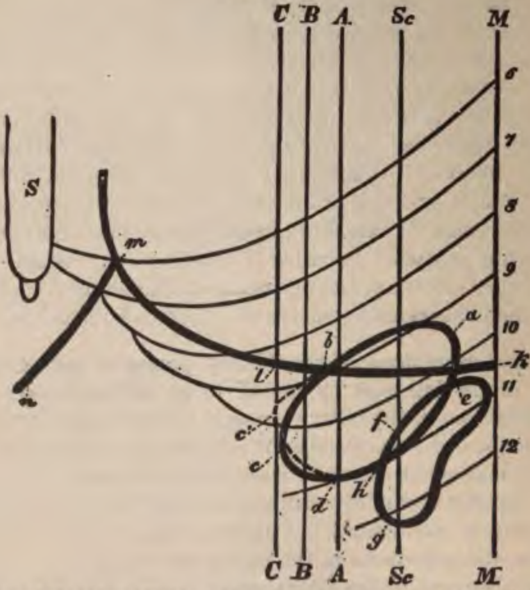
The diagnosis is often extremely difficult. The uncertainty is increased by the fact that calcular obstruction and malignant disease are sometimes associated. The detection of primary carcinoma elsewhere, or of metastatic nodules, indicates the existence of malignant disease. It was at one time thought that fever was wanting in malignant disease, but this is not an invariable rule.

THE SPLEEN.

What is the normal situation of the spleen, as determinable by physical examination?

The spleen (Fig. 31) is placed obliquely beneath the ribs, from

Fig. 31.



Relations of the spleen (Weil). *M*, middle line of back; *A*, *B*, *C*, axillary lines; *Sc*, line of scapula; *a b c d*, limits of spleen; *a b c' d*, limits of rhomboidal spleen; *e f g*, limits of kidney; *l b c*, angle between lung and spleen; *d h g*, angle between spleen and kidney; *n m*, lower margin of liver.

the ninth to the twelfth, on the left side below the axilla. The organ is about four inches long by three inches wide; its axis passes downwards and forwards. Posteriorly, the dulness of the spleen merges with that of the left kidney.

Floating Spleen.

To what symptoms does a floating spleen give rise?

The attachments of the spleen may become relaxed and prolonged, so that the mobility of the organ is abnormally increased, and its displacement is facilitated. The essential points in the recognition of a floating spleen are the presence of a solid body in an unusual situation, the absence of the spleen from its normal seat, the possible facility of replacement and the absence of pronounced symptoms.

Splenitis.

What are the symptoms of splenitis?

Inflammation of the spleen is a condition clinically obscure. It may occur in the course of infectious diseases or may be set up by emboli swept into the splenic vessels. It has in cases been attributed to traumatism. Under such circumstances the organ becomes enlarged. There are pain and tenderness in the left hypochondrium, or rather at the lower lateral aspect of the left chest, aggravated by respiration ; there are also nausea, vomiting and elevation of temperature. Inflammation of the spleen may terminate in suppuration. Then chills, fever and sweats are superadded. The greatest danger is from the rupture of an abscess into the peritoneal cavity.

THE PANCREAS.

Acute Pancreatitis.

What are the symptoms of acute pancreatitis?

The frequency of acute pancreatitis is not known, as the condition is difficult of recognition. It may result from extension of adjacent inflammation, from the irritation of matters contained in the blood, from traumatism and from hemorrhage into the pancreas. It is characterized by acute, deeply-seated pain and tenderness in the epigastric region, or perhaps colicky pain, shooting to the back and shoulders; by anorexia, thirst, nausea, vomiting of a thin viscid liquid, sometimes of bile; by dyspnea, restlessness, anxiety and depression; by febrile symptoms; and usually by constipation—phenomena similar to those presented by peritonitis. In some cases there is diarrhea, the stools being thin, watery, and resembling saliva. Death is the usual termination. It may be sudden, with the phenomena of collapse.

THE GENITO-URINARY APPARATUS.

Examination of the urine is important not only when disease in the genito-urinary tract is suspected, but also in many other varied conditions.

The *constitution of the urine* may be quantitatively or qualitatively altered. The proportions of normal ingredients may be increased or diminished. Substances not normally found in the urine may under certain conditions be present.

The *quantity of urine* excreted by a healthy adult in twenty-four hours varies between forty and fifty ounces. The quantity is physiologically *increased* after the ingestion of large quantities of fluid and when the cutaneous, pulmonary and intestinal transpiration is diminished. The quantity is physiologically *diminished* under the reverse conditions.

The quantity of urine excreted is altered in many conditions of disease. It is increased in diabetes insipidus and diabetes mellitus, in chronic nephritis and, temporarily, in emotional states, including hysteria. The quantity of urine is diminished in acute nephritis, in the last stages of chronic nephritis, in lithemia, in renal insufficiency, in conditions characterized by feebleness of the heart, in Asiatic cholera, in yellow fever, in acute intestinal obstruction and in febrile conditions generally.

Urine is normally clear, transparent, of an amber color, without obtrusive odor, of acid reaction and a specific gravity varying from 1010 to 1030. The color is paler or darker, and the specific gravity lower or higher, as the quantity is increased or diminished.

The *transparency of the urine* is interfered with by the presence of matters rendered insoluble by an altered reaction of the urine (such as phosphates), or by a change of temperature (such as urates); by the presence of matters foreign to normal urine (such as pus, chyle or blood); and sometimes as a result of the action of bacteria.

The *color of urine* is intensified when the quantity is diminished, as in fevers, or when the action of the skin is increased. The color is altered by the ingestion of some medicaments and foods and by the presence of other abnormal ingredients. Thus, rhubarb imparts a gamboge-yellow color to acid urine, a violet-red to alkaline urine; santonin imparts a golden-yellow to acid urine, an orange-yellow to alkaline urine; senna imparts a brownish, logwood a reddish, carbolic and gallic acids each a blackish tinge. Urates may color the urine orange-red. The presence of blood gives rise to a smoky, reddish or chocolate color; the presence of biliary pigment to a brownish or greenish hue; the presence of chyle to a milky appearance. In cases of melanotic tumors the urine may be dark or black; the color is likewise dark in some cases of pernicious anemia. The urine is pale when it is excreted in excess, as in hysteria, in diabetes and in chronic nephritis.

The *odor of normal urine* is typical, but not obtrusive. When fermentation takes place the urine becomes ammoniacal. The ingestion of turpentine imparts to the urine an odor of violets. Other aromatic oils impart odors abnormal to urine. The urine of diabetes mellitus has a sweetish odor. If it contain acetone, the odor resembles that of chloroform.

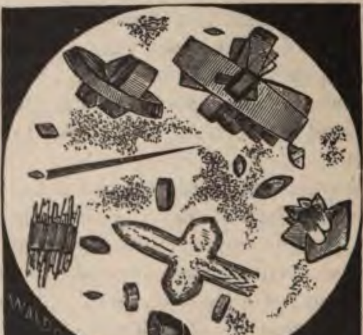
The total twenty-four hours' urine of a healthy person is of acid *reaction*. The urine may be alkaline from fixed alkali a few hours after a meal, especially if large quantities of alkalies have been taken; in cases in which the stomach is washed out; and in cases of obstinate vomiting. The urine is alkaline from volatile alkali when ammoniacal fermentation has taken place.

The *average specific gravity* of normal urine is 1018 or 1020. Except in diabetes mellitus, the specific gravity bears in general an inverse relation to the quantity of urine. It is heightened after the ingestion of large quantities of nitrogenous food, in diabetes mellitus and in acute nephritis and other febrile conditions. It is lowered in diabetes insipidus, in chronic nephritis, in hysteria, when the activity of the skin is lowered and after the ingestion of large quantities of fluid.

The normal average quantity of *urea* excreted in twenty-four hours is about five hundred grains. The excretion of urea is increased on a generous nitrogenous diet and in fevers in which the urine is not suppressed. The quantity of urea in the urine is diminished on a vegetable diet; when oxidation is interfered with, as in pulmonary or cardiac disease; in cases of grave hepatic disease, renal insufficiency or suppression of urine; and when the urea is retained in the circulation.

If urine or any fluid containing urea be concentrated to a syrupy consistence, by evaporation in a water-bath, and nitric acid be added, a crystalline precipitate of the rhombic plates of urea nitrate will be thrown down.

FIG. 32.



Uric acid and urates. (Funke.)

The quantity of *uric acid* excreted in the urine in twenty-four hours varies from eight to sixteen grains. It is increased when the diet is nitrogenous; when oxidation is defective, as in pulmonary or cardiac disease; after an attack of gout or lithema; and in febrile processes. It is diminished on a diet of carbohydrates; in chronic diseases after hemorrhage; during an attack of gout or lithema.

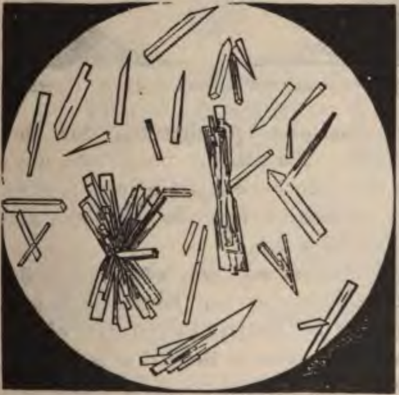
If ten parts of hydrochloric acid be added to one hundred parts of urine and the mixture be permitted to stand for forty-eight hours, a sediment of fine, red crystals of uric acid will form.

The quantity of *hippuric acid* excreted in the urine in twenty-four hours is between eight and thirty grains. It is increased on a vegetable, and diminished on an animal diet. It is only precipitated from acid urine, when it appears as rhombic crystals.

The quantity of *sodium chloride* excreted in the urine in twenty-four hours is about half an ounce. It depends upon the quantity ingested. The excretion of chlorides is diminished in febrile disorders, and especially when exudation or transudation occurs.

The presence of chlorides in the urine may be determined by first acidulating the urine with nitric acid, and then adding a solution of silver nitrate. If the precipitate that forms is dense and curdy, the quantity of chlorides is normal; if the precipitate is milky, the chlorides are diminished.

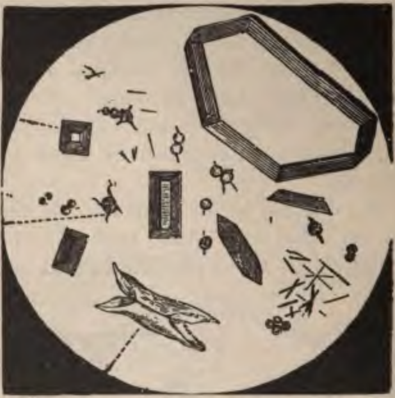
FIG. 33.



Calcium phosphate. (Laache.)

Between thirty and eighty grains of *phosphoric acid* are eliminated in the urine in twenty-four hours, two-thirds as alkaline (acid sodium and potassium) phosphates, and one-third as earthy (calcium and magnesium) phosphates. The quantity of phosphates excreted is diminished at the beginning of febrile processes, and increases with defervescence and convalescence.

Fig. 34.



Triple phosphates and ammonium urate. (Laache.)

The earthy phosphates are precipitated when the reaction of the urine is alkaline. The alkaline phosphates are precipitated by the addition of a solution containing one part each of magnesium sulphate, ammonium chloride and liquor ammoniæ, and eight parts of distilled water.

About thirty grains of *sulphuric acid* are eliminated as sulphates in the urine in twenty-four hours. The quantity varies with the character of the food and the degree of activity.

The presence of sulphates in the urine is determined by the addition of a solution of barium chloride, which gives a cloudy precipitate, insoluble in water or acids.

Biliary coloring matter appears in the urine when jaundice exists. The urine is yellowish, greenish or olive-brown in color, and stains the linen. When the jaundice is not due to biliary obstruction it is said that the biliary acids are not found in the urine.

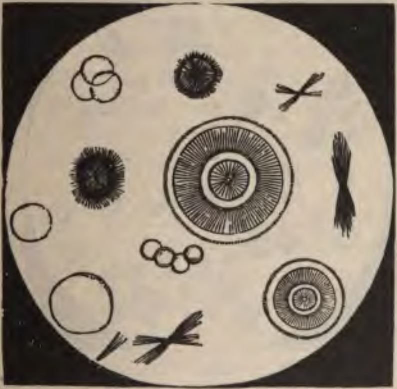
The presence of biliary coloring matter in the urine may be detected by overlaying nitric acid in a test-tube with urine, or by permitting the fusion of a drop each of nitric acid and of urine on a white plate. A play of color from green, blue, violet, red to yellow, results.

To detect the presence of the biliary acids in the urine a small quantity of cane-sugar is added to the urine, which is introduced into a test-tube, so as to overlay some sulphuric acid. A purple color is formed at the line of contact. If a bit of filter-paper is dipped in the saccharated urine and touched with a drop of sulphuric acid a purple color results.

What are leucin and tyrosin?

Leucin and *tyrosin* are products of the destructive metamorphosis of proteids, and are among the waste-products of pancreatic digestion. They do not occur in normal urine, but are found in the urine in cases of phosphorus-poisoning and of acute yellow atrophy of the liver, for which they may have diagnostic significance. They have also been exceptionally

FIG. 35.



Leucin and tyrosin. (Laache.)

noted in variola, typhus, pernicious anemia, and in a case of obstructive jaundice caused by hydatid cyst of the gall-duct.

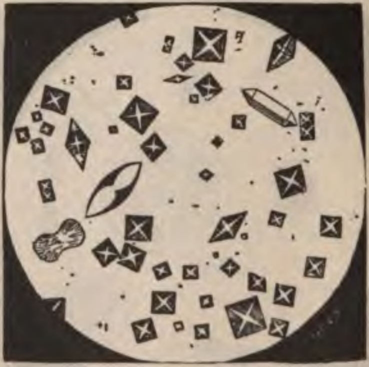
To obtain leucin and tyrosin in the sediment, the urine should be evaporated in a water-bath to syrupy consistence, or a drop may be boiled down on an object-glass. Leucin appears in the form of faintly shining spheres of variable size, the larger ones sometimes exhibiting radiation or concentric rings. Tyrosin crystallizes in very fine needles, commonly aggregated into sheaves or bundles.

Oxaluria.

What is oxaluria?

The presence of oxalates in the urine is sometimes associated with a complex of symptoms, to which the designation oxaluria has been applied. Individuals so affected complain of languor, of dull pains in the loins, are irritable or dejected, have boils or carbuncles; and their nutrition is impaired. The *urine*, the specific gravity of which is increased, contains an excess of urea and of oxalates, and occasionally a trace of albumin. The condition has been observed in persons in whom the nervous system has been severely put to task.

FIG. 36.



Calcium oxalate. (Laache.)

Pyuria.

What is pyuria?

The presence of pus in the urine constitutes *pyuria*. Pus appears in the urine when there is suppuration in any portion of the genito-urinary tract, as in urethritis, cystitis, ureteritis or pyelitis. Rupture of an abscess into the genito-urinary passages gives rise to the sudden discharge of a large quantity of pus. When, in women, leucorrhea exists, some of the pus may find its way into the receptacle for urine. The addition of caustic alkalies converts pus into a gelatinous viscid mass. Hydrogen dioxide gives a characteristic foaminess, from liberation of oxygen. The microscope is the most certain test. Urine containing pus responds to chemic tests for albumin.

When *urethritis* exists, micturition is burning. The first urine passed is turbid; that which follows may be clear. The reaction of the urine remains unchanged. In the vast majority of cases, urethritis is gonorrheal.

When there is *cystitis*, there may be vesical tenesmus, with frequent passage of small quantities of urine that is usually of alkaline reaction. Cystitis may be secondary to urethritis; it may be a result of obstruction, as from stricture of the urethra or an enlarged prostate; it may be associated with a calculus in the bladder; it may have been caused by the introduction of septic matters into the bladder in the course of some surgical procedure, such as the employment of an unclean catheter, sound or bougie; it may be tuberculosis; it is often seen in association with diseases of the brain and spinal cord. The urine is not only turbid, but it is usually also alkaline in reaction and offensive in odor, from ammoniacal fermentation.

Ureteritis and *pyelitis* are usually associated. Both are commonly secondary to cystitis, though they may result from the presence of calculi. In uncomplicated cases the urine is acid in reaction.

When an *abscess* ruptures into the genito-urinary tract, a certain sense of relief of tension is perceived, followed by the evacuation of a large quantity of pus in the urine, which had previously presented no abnormal characters.

Albuminuria.

What is albuminuria?

Albumin is found in the urine under many varied conditions; thus, urine containing pus or blood will respond to the tests for albumin. Under other circumstances, however, the albumin is derived directly from the circulating blood, as a result of the defective action of the epithelium of the secretory tubes of the kidney. Such a condition is present in the various morbid states of the kidney, in the course of fevers and infectious processes and in association with violent convulsions, obstructive disease of the heart, and interference with the respiratory function. The ordinary form of albumin found in the urine is serum-albumin; less commonly paraglobulin is present.

How is the presence of albumin in the urine to be determined?

The best test for the detection of albumin in the urine is that by heat. The urine should be clear and of acid reaction. If not clear, it should be filtered. If not acid, a drop or two of acetic acid—sufficient to impart an acid reaction, should be added. The upper half of the urine, in a test-tube, is heated. A resulting cloudiness is due to the presence of albumin or of phosphates. If the urine have been acid, the cloudiness is not likely to be due to phosphates. The addition of a few drops of acetic acid removes any doubt; if the cloudiness is dependent upon the presence of phosphates, it at once clears; if due to the presence of albumin, it persists.

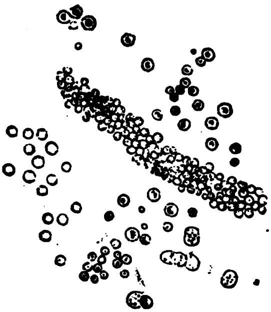
A simpler test for the presence of albumin consists in overlaying a quantity of nitric acid in a test-tube with the urine. A white ring at the line of contact of the two fluids is indicative of the presence of albumin. A faint ring of urates that forms above the line of contact is dissipated by the application of heat.

A saturated solution of picric acid used by the contact-method in the same way as nitric acid, the urine, however, being overlaid by the acid, constitutes a most delicate test for the detection of albumin in the urine, but it is equivocal, as it reacts with peptones, urates and with the alkaloids. Heat, it is true, dissipates the ring formed with these, but it may also cause a diffusion of the turbidity produced by albumin, so that the latter may escape detection.

What is the significance of the presence of tube-casts in the urine?

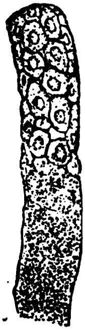
The presence in the urine of casts of the uriniferous tubules is indicative of nephritis. The presence of albumin is usually associated with that of tube-casts.

FIG. 37.



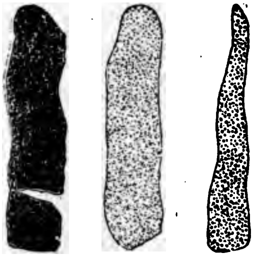
Red blood-corpuscles and a blood-cast of a uriniferous tubule. (Eichhorst.)

FIG. 38.



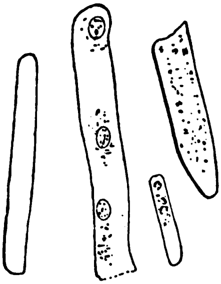
Epithelial cast of a uriniferous tubule. (V. Jaksch.)

FIG. 39.



Granular casts of the uriniferous tubules. (V. Jaksch.)

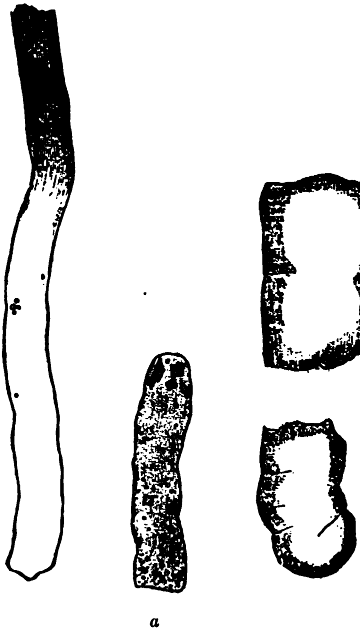
FIG. 40.



Hyaline casts of the uriniferous tubules. (Vierordt.)

The character of the casts varies according to the form of nephritis. *Blood-casts* and *epithelial casts* are indicative of an acute process; *granular casts* of a chronic process; *hyaline casts* may be present in many conditions. *Fatty casts* bespeak the late stage of a chronic parenchymatous nephritis—fatty kidney; *waxy casts* may be found in acute and chronic nephritis and in amyloid disease of the kidney.

FIG. 41.



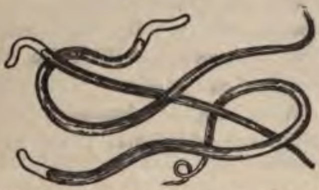
Waxy casts of the uriniferous tubules. *a*, a waxy cast containing crystals of calcium oxalate. (V. Jaksch.)

Chyluria.

What is chyluria?

The presence in the urine of chyle or its molecular base, fat, constitutes *chyluria.* The urine presents a whitish, milky appearance and displays a tendency to spontaneous coagulation. Albumin is present; in cases due to filaria, blood may be found. Under the microscope lymph-corpuscles and a finely granular basis are recognized. Chyle finds its way into the urine as a result of abnormal communication between the lymphatic system and the genito-urinary tract, in consequence of rupture following obstruction in the lymph-channels, most commonly by *filaria sanguinis hominis.* The scrotum and the lower extremities are sometimes infiltrated, and may be enlarged so as to create a resemblance to elephantiasis. *Filariosis* is a chronic affection, as a rule irregularly intermittent in its manifestations. When filariæ are present, ova or embryos should be discovered in the urine or in the blood obtained during the period of sleep or rest.

Fig. 42.



Filaria sanguinis hominis. (V. Jaksch.)

Lipuria.

What is lipuria?

The presence of fat in the urine constitutes *lipuria.* It may occur in health after excessive ingestion of fatty food. It has been observed in connection with fatty degeneration of the kidney, pyonephrosis, diabetes mellitus, phosphorus-poisoning and pregnancy. Urine containing chyle responds to the tests for fat. The condition may be recognized by the addition to the urine of a small quantity of potassium hydrate and then shaking with ether. The ether is permitted to evaporate and the fat collects as globules.

Hematuria.

What is hematuria?

The presence of blood in the urine constitutes *hematuria.* The *urine* is turbid and darker in color than normal; the specific gravity is increased; and albumin is present in considerable quantity. With the microscope there is no difficulty in distinguishing the red corpuscles.

Blood may be present in the urine as a result of hemorrhage from traumatism or ulcerative processes; in the course of acute inflammations in the genito-urinary tract; as a symptom of certain nervous diseases; as a manifestation of a hemorrhagic diathesis, as in leukemia, scurvy, purpura, hemophilia, exophthalmic goiter, and the like; as a manifestation of influenza or malaria, or as the result of drug-action. Like chyluria, hematuria may be due to *Filaria sanguinis hominis. Distoma hematobium* and *strongylus gigas* are additional parasites that may cause hematuria.

It may sometimes be important to determine whether the blood present in urine comes from the bladder or from the kidneys. A comparatively small quantity of blood; its uniform admixture with the urine; the presence of blood-casts of the tubules, or of the ureter; small size, discoloration, or so-called "ringing" of the blood-cells, point to renal hemorrhage. The passage of pure blood or of clots unmixed with urine, or with but slight admixture, or only at the beginning or close of urination, points to hemorrhage from the bladder or urethra. Exploration or cystoscopic examination may detect the source of hemorrhage and perhaps its cause, as a calculus or a neoplasm.

Hemoglobinuria.

What is hemoglobinuria?

When the coloring-matter of the blood appears in the urine the condition is designated *hemoglobinuria.* The coloring-matter of the blood may appear free in the urine as a result of conditions attended with disorganization of the blood, such as scurvy, purpura, pyemia, typhus, insolation, extensive burns, and poisoning by potassium chlorate, hydrogen arsenide, phosphorus, carbolic acid and chloral. Spontaneous hemoglobinuria has been observed in the new-born infant.

The *urine* is dark-red or chocolate-brown in color, of high specific gravity, and contains considerable albumin. Microscopically, few or no blood-cells are found. A drop of such urine treated on a slide with sodium chloride, acetic acid, and heat yields microscopically the flat, rhombic, prismatic tables known as Teichmann's crystals. The addition of a few drops of fresh tincture of guaiac to a small quantity of the same urine gives rise to a whitish or greenish coloration; shaken well, the addition of a few drops of a solution of hydrogen dioxide or of old oil of turpentine causes a change of color to blue. Spectroscopic examination reveals characteristic striæ.

The *attack* sets in with a rigor, to which succeed vomiting, diarrhea, collapse, cyanosis, stupor, and, usually, a fatal issue.

Paroxysmal Hemoglobinuria.

What is paroxysmal hemoglobinuria?

Hemoglobinuria may be *intermittent* and *paroxysmal*. In some cases no cause for the condition can be elicited; in most, however, the exciting factor has been exposure to unusual cold. Attacks may be induced by mental or physical exhaustion. The disease is most common in young adult males. The onset of a paroxysm is indicated by a sense of languor and fatigue, to which is soon added a feeling of chilliness. The fingers and toes and tips of the ears become numb, cold and cyanotic. The paroxysm ends by the restoration of a comfortable sense of warmth. During the attack, or a short time subsequently, the symptom that gives the disease its name appears. The urine, however, soon assumes a normal condition.

How are paroxysmal hemoglobinuria and malarial hematuria to be differentiated?

Paroxysmal hemoglobinuria is not known to be related to malarial infection; other possible manifestations of malaria are wanting; the periodicity of the attack is not rhythmical; malarial hematuria is a manifestation of profound malarial intoxication, associated with other distinct characteristics of malaria. In hematuria, with the microscope, many red blood-corpuscles are to be seen in the urine; the absence of blood-corpuscles in the urine is characteristic of hemoglobinuria. The one condition yields to treatment with quinine; the other does not. Examination of the blood in a case of paroxysmal hemoglobinuria discloses nothing characteristic; an examination of the blood in cases of malarial hematuria reveals the presence of characteristic parasites.

Endemic Hematuria.

What is endemic hematuria?

In tropical climates the presence of a nematode worm, the *distoma hematobium* or *Bilharzia hematobia*, in the veins about the bladder, occasions the appearance of blood in the urine. The ova of the parasite find their way into the genito-urinary passages and there give rise to irritation and inflammation and extravasation of blood. The disease is most common in youth; it shows a tendency to subside after the age of puberty. The condition predisposes to the development of urinary calculi. The *diagnosis* depends upon the endemic character of the affection and the detection of ova, which are often concealed in shreds of tissue contained in the urine.

Diabetes Insipidus.

What are the clinical features of diabetes insipidus or polyuria?

Diabetes insipidus or *polyuria* is a morbid condition characterized by the elimination of an excessive quantity of feebly acid urine, of low specific gravity, containing neither albumin nor sugar. There is increased thirst; an abnormal quantity of fluids may be ingested, than which, however, the quantity of urine excreted is usually in excess. The *skin* and visible mucous membranes may be rough and dry. In some cases the *appetite* is excessive. The general *nutrition* may be maintained, but is rather more likely to suffer deterioration, as manifested by wasting and debility. Diabetes insipidus is not directly fatal. Its *etiology* and *pathology* are obscure. In many cases, there is a history of injury to the head, or disease of the brain is found post-mortem. It may depend upon disease of the pancreas. Several cases have been found in a single family; in other instances, some members of a family present diabetes insipidus, others diabetes mellitus.

How are diabetes insipidus and chronic interstitial nephritis to be differentiated?

In cases of chronic interstitial nephritis the quantity of urine eliminated may be abnormally large, but it does not attain the proportions encountered in diabetes insipidus; in addition, careful investigation will detect the presence of albumin and casts in the urine, as well as the existence of other symptoms indicating a degenerative lesion of the kidney: heightened arterial tension, hypertrophy of the heart, breathlessness, edema, headache, diarrhea.

How are hysterical polyuria and diabetes insipidus to be differentiated?

The excessive discharge of urine that is sometimes observed in hysterical patients is paroxysmal rather than persistent; and does not constitute the essential feature of the disease, but is rather an incidental symptom of a malady of protean feature.

Diabetes Mellitus.

What are the clinical features of diabetes mellitus?

Diabetes mellitus, *glycosuria*, *or mellituria*, is a morbid condition attended by the discharge of large quantities of urine characterized by the persistent presence of a varying quantity of glucose. It is sometimes observed in connection with cerebral disease, sometimes in connection with disease of the pancreas or of the liver, sometimes in women at the menopause, but is commonly unassociated with any evident disease. In many cases there is a racial or family predisposition or a gouty or rheumatic diathesis. In not a few instances its advent has been preceded by grief, anxiety or profound emotion.

Among the earliest *symptoms* is a gradual failure of health, with a sense of fatigue disproportionately great. It may be observed that the patient is passing an excessive quantity of *urine*, examination of which shows it to be pale in color, of a sweetish, fragrant odor, of high specific gravity, and chemically containing glucose. The proportion of sugar varies, being increased by indulgence in and diminished by abstinence from sugars and starches. At the same time, there is increased thirst, together with the ingestion of large quantities of fluids. The *appetite* is also wont to be excessive and large quantities of food are taken. Constipation is the rule, though exhausting diarrhea may occur. The *skin*, mouth and throat are dry. A sweetish taste is perceived. The *breath* may exhale a fragrant, a balsamic, an ethereal odor, or an odor of chloroform. The *teeth* become carious, the gums spongy and loose. Boils and carbuncles are common. Persistent furunculosis, without obvious cause, should always awaken a suspicion of diabetes. In women, pruritus vulvæ, and, in men, excoriations about the meatus urinarius have been observed. Failure of sexual vigor is sometimes an early symptom. Emaciation and debility may ultimately become extreme. Exceptionally an appearance of perfect health is preserved. Some have distinguished two groups of cases, those with a tendency to emaciation, and those with a tendency to obesity.

Sometimes, there is sense of chilliness and the temperature is subnormal. Gangrene from vascular occlusion may result and bring about a fatal issue. After death the bladder is often found hypertrophied. Cataract not rarely develops in the course of diabetes. Nephritis is sometimes superadded. *Death* commonly results from an intercurrent affection, such as pulmonary tuberculosis or pneumonia. Sometimes coma appears, with or without delirium and convulsions. Coma is in many cases preceded for a few days by the appearance in the urine of acetone and diacetic acid, concurrently, it may be, with diminution of the percentage of sugar.

What is Moore's test for the presence of glucose in the urine?

The simplest test for the presence of sugar in the urine is Moore's test. To a moderate quantity of urine in a test-tube half as much liquor potassæ is added. The mixture is heated. If sugar be present in greater proportion than 0.3 per cent., a sherry-wine color results. If a drop or two of nitric acid be added, the brownish color disappears and the odor of molasses becomes perceptible. Other substances than sugar respond similarly in color; so that the test cannot be infallibly relied upon.

What is Boettger's test?

To a small quantity of urine in a test-tube is added about an equal quantity of liquor potassæ, and then a small pinch of bismuth subnitrate. The mixture is heated; if sugar be present a black precipitate of metallic bismuth is thrown down. Sulphur compounds precipitate black sulphides, so that albumin, if present, should first be removed by precipitation and filtration. If the reaction fails to take place, the presence of sugar in significant quantity can safely be excluded.

What is Trommer's test?

To a moderate quantity of urine in a test-tube a smaller quantity of liquor potassæ, and then a few drops of a ten per cent. solution of cupric sulphate are added. If heat be applied, an orange-red precipitate of cuprous oxide is thrown down if sugar be present.

What is Fehling's test?

Two solutions are used. One consists of an ounce of cupric sulphate and sufficient water to make fifteen ounces; the other of five ounces of crystallized Rochelle salts, three ounces of a solution of sodium hydrate having a specific gravity of 1.34, and sufficient water to make fifteen ounces. When the test is to be made, equal quantities of the two test-solutions are heated in a test-tube. The urine is then slowly added. Should no orange-red precipitate result, the presence of sugar can be excluded.

Fehling's test can be applied for quantitative analysis by employing solutions of which a known volume will be reduced by a definite quantity of glucose.

What is the fermentation-test?

If a bit of baker's yeast be added to urine containing sugar, fermentation takes place, carbon dioxide and alcohol being generated. In making a quantitative examination the specific gravity is taken and recorded; eight ounces of urine are introduced into a pint bottle, a piece of compressed yeast about the size of a walnut added, and the bottle closed by a perforated cork, and placed in a warm place (from 86° to 90° F.) for four or five hours or longer. The urine is then permitted to cool, and its specific gravity is again taken. Each degree of loss of specific gravity represents about one grain of sugar to the ounce, or the total number of degrees lost multiplied by 0.23 will give the percentage of sugar.

What are the tests for acetone?

1. To the urine is added a small quantity of yellow precipitate (mercuric oxide), freshly prepared by mixing an alcoholic solution of potassic hydrate with mercuric chloride. If acetone is present some of the mercurial will be dissolved. The urine is then filtered and the filtrate overlaid with ammonium sulphide. A black ring of mercuric sulphide shows that the reagent has been dissolved, and thus indicates acetone. Or, 2. About 4 c.c. (1 dram) of solution of potassic hydrate, containing 1 gram (15 grains) of potassium iodide, are placed in a test-tube and boiled. An equal volume of urine is then cautiously poured in, so as to float on the surface of the alkaline liquid. At the point of contact a ring of phosphates will form, which, if acetone be present, will in a few minutes be colored yellow and studded with crystals of iodoform.

What is a convenient test for diacetic acid?

Solution of ferric chloride is slowly added to the urine; the phosphates thus precipitated are removed by filtration, and more ferric chloride is added. If glacial acetic acid is present, the urine becomes wine-red. The test must then be repeated with urine previously boiled. If the boiled urine gives no reaction with the iron solution, or only a slight coloration, and if acetone is likewise found to be present, it may be concluded that *diaceturia*, a forerunner of diabetic coma, exists.

How are diabetes insipidus and diabetes mellitus to be differentiated?

The diagnostic distinction between diabetes mellitus and diabetes insipidus is the presence or absence of sugar in the urine, of which the specific gravity is lower in the latter than in the former. The occurrence of boils, of itching of the skin, of cataract and of gangrene, the presence of a sweetish taste and of a fragrant breath, encountered in saccharine diabetes, are wanting in insipid diabetes. The prognosis in the case of diabetes insipidus is more favorable than in that of diabetes mellitus.

Cystitis.

What are the symptoms of cystitis?

The mucous membrane of the bladder may undergo inflammation in the course of constitutional disorders, or as the result of local irritation or infection. Cystitis gives rise to pain in the hypogastrium, a frequent desire to urinate, followed by the evacuation of small quantities of urine, attended with burning pain. The last drops of urine passed are opaque and viscid. The quantity of urine voided does not in the aggregate exceed the normal. When the cystitis is acute the reaction of the secretion is acid; in chronic cystitis the urine is alkaline; the urine is turbid and precipitates a dense, copious sediment; it is albuminous in a degree proportionate to the quantity of pus contained. Examined microscopically the sediment is found to be made up of pus-corpuscles, pavement epithelial cells and mucus. In the earliest stages of acute cystitis the urine may contain blood; in chronic cystitis, crystals of the triple ammonio-magnesium phosphate may be found. The walls of the bladder become thickened, its cavity reduced in size, its lining membrane ribbed. The dangerous sequelæ of cystitis are ureteritis, pyelitis, hydronephrosis and suppurative nephritis, dependent upon extension of the inflammatory process.

How are cystitis and nephritis to be differentiated?

Pain in the hypogastrium is a feature when there is acute cystitis. If pain attend acute nephritis, it is referred to the loins. Urine is passed less frequently and in smaller quantities when acute nephritis exists than is the case in acute cystitis. The urine of cystitis is the more distinctly purulent; that of nephritis, the more distinctly albuminous. In cases of nephritis, the urine contains casts of the uriniferous tubules. The urine of simple cystitis never contains tube-casts. The alkalinity of the urine, the presence of pus, and the absence of tube-casts, in addition to the absence of other phenomena, such as edema, dyspnea, anemia, cardiac hypertrophy, distinguish chronic cystitis from chronic nephritis.

Vesical Calculus.

To what symptoms does a vesical calculus give rise?

The presence of a calculus in the bladder gives rise to irritation and inflammation. In addition to the signs of cystitis—frequent, burning discharge of small quantities of urine of alkaline or acid reaction—there occur attacks of vesical tenesmus, with complete obstruction to the discharge of urine, with pain referred to the glans penis, followed by the presence of blood in the urine. Careful examination, by means of a sound or the cystoscope, may furnish conclusive evidence. A vesical calculus may not only excite cystitis, but it may also give rise to inflammation and dilatation of the ureters and pelves of the kidneys and to hydronephrosis.

Neoplasms of the Bladder.

To what symptoms do neoplasms of the bladder give rise?

When the lining of the bladder is the seat of new-growths, hemorrhage is of frequent occurrence, together with paroxysms of vesical tenesmus. The *urine* usually contains cellular evidence of the presence of a neoplasm, while cystoscopic examination may afford ocular demonstration of the fact.

Pyelitis.

What are the symptoms of pyelitis?

Inflammation of the mucous membrane of the pelvis of the kidney may arise in the course of acute infectious diseases, may result by extension from the urethra, through the bladder and ureters, or from the presence of a calculus or a new-growth. Pyelitis may be tuberculous. There is pain in the loin. The *urine*, the reaction of which remains acid, precipitates a copious sediment principally constituted of pus, with some epithelial cells. From time to time the urine may contain blood.

In cases of tuberculosis, tubercle-bacilli may be found in the urine, and there may be evidences of tuberculous disease elsewhere. It is said that bacilli morphologically indistinguishable from tubercle-bacilli and presenting the same color-reactions are sometimes found in the urine of healthy persons. The numbers are smaller, and the organisms are less constantly present than in cases of tuberculosis, and the inoculation-test proves them to be innocuous.

How are pyelitis and cystitis to be differentiated?

The pain of pyelitis is referred to the lumbar region ; that of cystitis to the hypogastric region. Small quantities of urine are frequently evacuated in case of cystitis ; neither quantity nor frequency are altered in uncomplicated pyelitis. When there is cystitis, the urine is most likely to be alkaline in reaction ; when there is pyelitis, it is usually acid.

Renal Inadequacy.

What is renal inadequacy?

Some individuals never pass more than a normal or slightly less than normal quantity of urine of low specific gravity and containing abnormally little urea, even though they should partake of an excess of fluids. The condition has been termed *renal inadequacy*. The symptoms are vague and indefinite. There is a sense of ill-being, an undue readiness of fatigue, and a lowered resistance to disease. Patients so affected bear operative interference badly. There may be slight edema, but the urine contains neither albumin nor casts.

Floating Kidney.

What are the symptoms of a floating kidney?

The kidney may be furnished with a mesentery, attached to which it may find its way into the abdominal cavity, without necessarily giving rise to significant symptoms. The condition is to be recognized by the mobility, the possibility of replacement, a void in the usual situation of the kidney, and the fact of the remaining viscera occupying their proper positions. By twisting of the ureter, urine may accumulate in the renal pelvis, to be evacuated when the quantity has reached a certain limit. In addition, there may be a sense of weight and dragging in the abdomen, with pain usually dull, but occasionally acute and associated with tenderness. Sometimes the distress is more aggravated, and nausea and vomiting occur. Floating kidney is more common in females than in males; the right kidney is more frequently affected than the left.

Renal Calculus.

To what symptoms does a renal calculus give rise?

A calculus in the pelvis of the kidney occasions constant dull pain in the loin, aggravated into great intensity in paroxysms, superinduced by active physical exercise or by concussion. Following the paroxysm the urine contains blood. In the interval the urine contains pus. If the calculus enter the ureter, efforts at its expulsion give rise to excruciating pain in the course of the ureter, with retraction of the corresponding testicle, numbness of the thigh, nausea and vomiting. When a renal calculus is a cause of obstruction to the flow of urine, hydronephrosis or atrophy of the kidney may result.

Acute Nephritis.

What are the clinical features of acute nephritis?

Acute inflammation of the kidney is common in association with acute infectious diseases—especially scarlatina, erysipelas, small-pox and yellow fever. It may follow exposure to cold. It sometimes develops in the course of pregnancy. The kidneys are large and smooth and reddened. The inflammation particularly involves the parenchyma of the organs. The occurrence of acute nephritis may be manifested by a chill, which is followed by fever, by pains in the loins, by headache, perhaps with nausea, vomiting and diarrhea. The *face* (notably the eyelids) is puffy; the extremities are swollen and edematous. The excretion of *urine* is diminished to a minimum; there may be actual suppression. The urine presents a smoky, reddish, turbid aspect; its specific gravity is high; it is deficient in urea, but it contains a decided quantity of albumin and hyaline and epithelial casts, and blood-casts, as well as free blood-corpuscles. The symptoms of *uremia* may supervene; to the headache, nausea and vomiting, are added derangement of vision, delirium, convulsions and coma. In the course of several weeks, the symptoms gradually subside, though the accompanying anemia yields but obstinately, while the urine may continue albuminous for a long time, perhaps permanently. Inflammations of the serous membranes are frequent in the course of acute nephritis.

Chronic Parenchymatous Nephritis.

What are the symptoms of chronic parenchymatous nephritis?

Recovery from acute inflammation of the kidney may be imperfect; repeated attacks may leave a permanently damaged organ; finally an inflammation of the parenchyma of the kidney may be insidious and essentially chronic from the outset. However produced, the *symptoms* are variable. The essential characteristic is the persistent presence of a considerable quantity of albumin in the urine, together with granular, hyaline or fatty tube-casts. The quantity of urine may scarcely deviate from the normal. A characteristic variety of retinitis may be detected with the ophthalmoscope. Edema of the face and extremities occurs, anemia is manifest, assimilation is impaired, the arterial tension is heightened, and the heart becomes enlarged. Ascites is common. Edema of the larynx or lungs, or effusions into the pleuræ or pericardium may take place suddenly or gradually. Headache, impaired vision, somnolence, convulsions and coma, from uremia, may develop. Individuals affected with chronic parenchymatous nephritis badly resist disease. The probabilities are that the so-called fatty kidney is but a modification of the kidney of chronic parenchymatous nephritis, dependent upon secondary degeneration. The symptoms of both conditions are much alike. In the secondary degeneration, the presence of fatty casts and of oil-globules is characteristic.

Amyloid Kidney.

What are the clinical features of amyloid disease of the kidney?

Amyloid, *lardaceous* or *waxy disease of the kidney* depends upon a peculiar hyaline degeneration of the coats of the renal capillaries, which is often associated with some degree of chronic parenchymatous nephritis, and with amyloid degeneration in other organs, such as the liver, the spleen and the intestine. The *symptoms* are practically those of the associated condition. In addition to edema, albuminuria, the presence of tube-casts in the urine, heightened arterial tension and cardiac hypertrophy, there may be a peculiarly waxy complexion, diarrhea, and painless enlargement of the liver, without ascites and jaundice. Amyloid degeneration results usually in consequence of protracted suppuration or ulceration, or of syphilis.

Chronic Interstitial Nephritis.

What are the symptoms of chronic interstitial nephritis?

The interstitial structure of the kidney participitates, in some degree, in every inflammation of the organ. The involvement becomes especially obvious in the late stages of all forms of nephritis. In some cases, however, the interstitial connective tissue undergoes hyperplasia from the outset, ultimately contracting and giving rise to a diminution in the size of the organ, which becomes small, red, granular ; the capsule is thickened ; on attempting to remove it the adherent cortex tears. This chronic interstitial nephritis is often but one part of a widely distributed arterio-capillary fibrosis. The principal *symptom* referable to the kidneys is frequent micturition, the urine being considerably increased in quantity, of a low specific gravity, and containing a trace of albumin and hyaline and granular tube-casts. Albumin and casts are sometimes only to be found by repeated, careful examination. There is little or no edema, unless dilatation of the heart develop. There is shortness of breath. The arterial tension is high and the heart is hypertrophied. The second sound of the heart is accentuated. There is sometimes persistent diarrhea. Retinitis, papillitis, and hemorrhages may occur. *Death* usually results from uremia or cerebral hemorrhage. The commonest causes of chronic interstitial nephritis are alcoholism, gout and lead-poisoning.

What are the symptoms of uremia?

Uremia is an auto-intoxication, which sometimes arises in the course of disease of the kidneys and is dependent upon the retention in the fluids of the body of certain products that it is the function of the kidneys to remove. Uremia gives rise to headache, vertigo, nausea, vomiting, diarrhea, dilatation of the pupil, altered vision, delirium, somnolence, convulsions and coma. Effusions frequently accompany the condition. The temperature may be subnormal. It has exceptionally been observed to be febrile. The body and breath exhale a musty odor. Uremia is one of the gravest complications of disease of the kidneys.

How are a uremic seizure and a paroxysm of epilepsy to be differentiated?

Probably dependent upon analogous conditions of irritation, there is little difference between the convulsions. The onset of an epileptic attack is sudden, attended with a cry and biting of the tongue, and possibly preceded by an aura; the convulsions of uremia are usually preceded by headache, vertigo and nausea, and occur without sound or injury to the tongue. The urine may contain albumin during or after an epileptic paroxysm, but casts are present as well when the convulsions are uremic. Epileptic seizures are far more likely than uremic convulsions to have been and to be repeated. A knowledge of the previous history of the case simplifies the diagnosis.

How are uremia and insolation to be differentiated?

The failure on the part of the skin as well as on the part of the kidneys to eliminate certain excrementitious products may in cases of insolation give rise to phenomena resembling those presented by uremia; but in the former the temperature attains a height never seen in the latter, while the season of the year at which the accident happens, as well as the attendant circumstances, makes the diagnosis clear.

Abscess of the Kidney.

What are the symptoms of abscess of the kidney?

Suppuration may take place in the kidney as the result of an acute infectious nephritis, as a part of a general pyemia, or in consequence of traumatism. When pus has formed, the *symptoms* occasioned are those to which imprisoned pus usually gives rise: rigors, hectic fever, sweats, emaciation. The *urinary secretion* will be altered in accordance with the intensity and extent of the causative or associated nephritis. In the urine may be found albumin, pus, tube-casts and blood-corpuscles. There may be pain and tenderness in the region of the kidney. If the accumulation of pus attains considerable proportions it may give rise to a palpable tumor, with fluctuation, in the loin. The abscess may rupture into the pelvis of the kidney, into an adjacent viscus, into the abdominal cavity; or the pus may make its way out through the abdominal wall.

Perirenal Abscess.

What are the clinical features of perirenal abscess?

Suppuration may take place in the perirenal connective tissue as a result of an extension of inflammation in the pelvis or in the structure of the kidney or as a result of blows or injuries. The condition is to be recognized by the existence of pain and tenderness in connection with a boggy, fluctuating tumor in the loin, associated with chills, fever, sweats and emaciation. The tumor is not movable, and cannot be separated from the kidney. Exploratory puncture may detect the presence of pus. Such an abscess may burrow and find an exit above or below Poupart's ligament, in the loin, into the kidney, ureter or bladder, into the peritoneum, intestines or into a lung.

How are a perinephric and a renal abscess to be differentiated?

An abscess in the loin developing in the course of pyemia is more likely to be renal than perinephric; an abscess following traumatism is more likely to be perinephric than renal; while disease of the pelvis or of the kidney may give rise to one or the other. The pus of a perinephric abscess is more likely than that of a renal abscess to burrow and point at some distant spot. In cases of renal abscess evidences of disease are likely to appear in the urine; there may be pus with albumin and tube-casts.

Tuberculosis of the Kidney.

What are the clinical features of tuberculosis of the kidney?

Miliary tubercles may be developed in the kidneys in common with other organs, as a part of an acute miliary tuberculosis. More usually, when the kidney is tuberculous, the foci are more extensive, and exhibit a tendency to undergo caseation. The kidney is rarely the exclusive seat of tuberculosis. As a rule, other portions of the genito-urinary tract are likewise involved, and not uncommonly the lungs as well. Miliary tuberculosis of the kidney does not give rise to symptoms apart from those of the general condition of which it is but an incident. In the more localized lesion, the symptoms are vague. There may be pain in the loin, with tenderness; exceptionally a tumor is discoverable on physical examination; the urine may contain pus and detritus, in which tubercle-bacilli can, perhaps, be found; sometimes there is hematuria; there may be hectic fever. Ultimately emaciation sets in, exhausting sweats take place and the vitality fails. Tuberculosis of the kidney is more frequent in the young than in the old, in males than in females.

Malignant Disease of the Kidney.

What are the clinical manifestations of malignant disease of the kidney?

Malignant disease of the kidney is sarcomatous or carcinomatous. Sarcoma may be congenital or it may appear during childhood. Carcinoma rarely develops before forty. The classical symptoms are tumor, pain and hematuria.

The swelling occupies one of the loins, and is separated from the anterior abdominal wall by the large intestine. The pain is dull and heavy, with acute exacerbations. Hematuria is not constant. Its absence is of less significance than would be its presence. In addition, the appetite fails; there may be nausea or vomiting, constipation or diarrhea; emaciation sets in; and cachexia develops. Edema of the lower extremities and ascites may result from pressure on the veins.

Hydronephrosis.

What is hydronephrosis?

When there exists at any part of the genito-urinary tract an obstruction to the flow of urine, such as may be occasioned by a calculus, a neoplasm, an enlarged prostate gland, a kink or a twist in, or pressure from without upon, a ureter, fluid accumulates behind the point of obstruction, and causes dilatation. The condition is designated hydronephrosis. It may be unilateral or bilateral. If it be the urethra or the orifice of the bladder that is obstructed, the bladder, ureters, and pelves of the kidneys all become thickened and dilated. If but one ureter is obstructed the corresponding pelvis suffers. The dilatation of the pelvis gives rise to compression and atrophy of the renal parenchyma. Sometimes the fluid resembles pale urine of low specific gravity ; at other times it contains pus. In the latter case the condition is designated *pyonephrosis.* The symptoms are ill-defined. There may be dull pain, a sense of fulness, and a fluctuant tumor in one or the other loin, attended perhaps with the periodic discharge of large quantities of urine, as the obstruction is temporarily relieved, with a corresponding recession of the symptoms. There may, in addition, be rigors, hectic fever, sweats and emaciation. Constipation due to pressure upon the bowel, is not uncommon in cases of hydronephrosis. The urine may be normal, except at such times as the periodic copious discharges take place, when pus or other matters may be found present. In cases of double hydronephrosis there is great liability to the sudden development of uremia.

Hydatid Cyst of the Kidney.

What are the symptoms occasioned by an hydatid cyst of the kidney?

The kidneys are relatively a common seat of hydatid cysts. Such a cyst in connection with the kidney may give rise to a fluctuating tumor in the loin or in the lateral region of the abdomen, yielding the characteristic thrill on percussion, and occasioning symptoms dependent upon pressure ; or prior to surgical exploration, it may not give rise to definite symptoms and may elude physical exploration. Under any circumstances a diagnosis may not be possible, unless the contents of the cyst find their way into the genito-urinary tract and are evacuated ; when the detection of the hooklets in the urine leaves no room for doubt. The recognition of an additional cyst in another situation may facilitate the diagnosis.

How are an hydatid cyst of the kidney and hydronephrosis to be differentiated?

An hydatid cyst of the kidney and hydronephrosis both occasion a fluctuating renal tumor, but the peculiar thrill on percussion that characterizes many hydatid cysts is wanting in all cases of water-logged kidney. Hydronephrosis is dependent upon some form of genito-urinary obstruction; such a condition bears no relation to the development of an hydatid cyst. The periodic discharge of a large quantity of fluid, with a temporary disappearance of the tumor, is characteristic of hydronephrosis; should a discharge of fluid take place, in connection with an hydatid cyst, microscopic examination will reveal the characteristic hooklets in the urine.

THE NERVOUS SYSTEM.

How is a study of diseases of the nervous system to be approached?

By an investigation of motion, sensation, reflex action, nutrition, electric reaction and cerebral functions.

How may motion be affected?

The power of muscular movement may be impaired or lost; motility may be exaggerated; or coördination may be impaired. Complete loss of muscular power is called *paralysis;* partial loss, *paresis.* Motor over-action is seen in spasm and convulsion. Incoördination gives rise to *ataxia.*

The power of voluntary motion is impaired or lost in destructive disease at any part of the motor tract, from the cerebral cortex to the end-plates. Movement may be interfered with by disease of the muscle itself, or of an adjacent joint; or abstained from on account of the pain that it causes in a joint or muscles.

Loss of motor power confined to one side of the body is called *hemiplegia;* if bilateral, *diplegia;* limited to the lower extremities, *paraplegia;* localized to a member, *monoplegia.*

Hemiplegia is usually dependent upon a unilateral cerebral lesion; it may possibly be caused by a unilateral lesion seated in the upper portion of the cord.

Diplegia may be a result of symmetrical bilateral cerebral lesions, or of a lesion in a situation where the motor tracts are close together; like hemiplegia, it may possibly be caused by a lesion in the upper portion of the spinal cord.

Paraplegia is invariably a result of disease of the cord.

Monoplegia is a result of a circumscribed lesion of the brain, usually in or near the motor cortex. Any form of paralysis may be simulated by hysteria.

Spasm or *convulsion* is indicative of irritation at any part of the motor tract. It may occur in acute disease, cerebral (hemorrhage, embolism, thrombosis) or constitutional (tetanus, hydrophobia) ; in chronic irritative disease (cortical softening, tumor) ; or it may be a symptom of nutritional disease (epilepsy), or of functional disease (hysteria) ; it may also occur in disease not strictly nervous (uremia).

Ataxia is the result of derangement of the conducting fibers or the coördinating centers in the cord or cerebellum. Loss of tactile sensation also gives rise to ataxia. Defective equilibration may result from disease of the internal auditory apparatus.

What are the varieties of sensation?

Common or tactile sensibility, the pain-sense, the temperature-sense, the muscular-sense ; special sensation : smell, vision, taste and hearing.

How may sensation be affected?

Sensation may be impaired, retarded, lost, perverted or heightened. Impairment of sensation is *hypesthesia;* loss of sensation is *anesthesia;* heightening of sensation, *hyperesthesia;* perversion of sensation, *paresthesia.* Alterations in sensation are dependent upon derangement in the course of the sensory path. As the lesion may be destructive or irritative, so may the symptoms be those of diminished sensation, of supersensibility, or of perverted sensation. One or the other variety of sensation may be affected without involvement of all.

What are the varieties of reflexes?

Superficial or cutaneous, and muscular or deep.

What are the principal superficial reflexes

The plantar, the gluteal, the cremasteric, the abdominal, the epigastric, the conjunctival, the reaction of the iris to light, and to cutaneous irritation.

What are the principal varieties of deep reflexes?

The knee-jerk, ankle-clonus and the elbow-jerk.

How are the reflexes altered in disease?

They may be exaggerated, enfeebled or entirely lost.

The superficial reflexes are exaggerated when the influence of the inhibitory apparatus is removed, or when the activity of the reflex apparatus of the spinal cord is heightened; the deep reflexes are exaggerated under similar conditions, and likewise when degeneration has taken place in the cerebro-spinal motor tract. Both superficial and deep reflexes are lost when the reflex arc is interrupted at any point in its course—sensory nerve, reflex center in the cord, and motor nerve.

How is the nutrition affected by disease of the nervous system?

Trophic changes often attend disease of the nervous system. Bedsores sometimes form. Muscles may waste. A paralyzed limb is edematous and its temperature is subnormal; in children the growth of paralyzed parts is retarded. Decided alterations in joints may take place. The skin sometimes becomes glossy. Trophic changes are most marked in degenerative disease of the nerves and of the gray matter of the cord, especially of the anterior horns. The nutrition of a palsied member slowly suffers.

How are the electric reactions affected by disease?

The normal response of muscle and nerve to both the voltaic and faradic currents may be enfeebled or exaggerated—a *quantitative change;* or the response of nerve to both faradism and voltaism, and of muscle to faradism may be enfeebled or lost, while the response of muscle to voltaism persists and may be exaggerated and is altered in its polar relations—a *qualitative change*, the *reaction of degeneration.*

Normally, the response of muscle to voltaic stimulation is most ready upon application of the kathode and making (closure) of the current; least ready upon application of the kathode and breaking (opening) of the current. Response upon anodal closure and anodal opening is less ready than that of kathodal closure, more ready than that of kathodal opening.

The sequence: (1) K.Cl.C.; (2) $\begin{matrix}\text{An.O.C.;}\\ \text{An.Cl.C.;}\end{matrix}$ (3) K.O.C.; is called the normal formula. A deviation from this sequence gives a formula expressive of the *reaction of degeneration*—a *qualitative* change. Quantitative changes take place in simple wasting dependent upon disuse or disease of the muscle or of an adjacent joint, or upon interruption in the motor path between the cerebral cortex and the ganglion-cells of the anterior horns of the spinal cord; qualitative changes take place in degenerative muscular wasting dependent upon lesions of nerve or cord, between the motor end-plates and the ganglion-cells of the anterior horns inclusive.

How are the cerebral functions affected by disease of the nervous system?

Delirium, headache and vertigo may appear. Vision, hearing, taste or smell may be perverted, excessively acute, impaired or lost; memory may be enfeebled; speech may be altered or lost; articulation may be defective; the emotions may be affected; consciousness may be deranged; the higher mental powers may suffer deterioration.

Neuritis.

How may neuritis be classified?

Into perineuritis, interstitial neuritis and parenchymatous neuritis. Neuritis may be acute or chronic; it may affect one nerve or many.

What are the causes of neuritis?

Inflammation of a nerve may result from local injury or compression, from adjacent inflammation, from exposure to cold, or from the influence of irritating matters circulating in the blood. Injury, compression, adjacent inflammation and exposure to cold give rise to circumscribed neuritis; irritants in the blood to multiple neuritis.

What are the symptoms of neuritis?

When a nerve is inflamed, hyperesthesia, paresthesia and anesthesia, pain, tingling, numbness in the peripheral distribution and tenderness in the course of the nerve-trunk in turn appear, with impairment or loss of motion. If the exciting conditions be maintained, nerves degenerate and muscles waste and the skin becomes glossy. Exposure to cold is a cause of inflammation of the facial nerve; rheumatism, of inflammation of the sciatic; lead-poisoning, of inflammation of branches of the radial, median and ulnar; pressure, of inflammation of the musculo-spiral; syphilis and alcoholism, of inflammation of the peripheral nerves of the extremities. Obstinate neuralgias are sometimes dependent upon neuritis. Acute neuritis is attended with fever and other constitutional phenomena.

With what conditions may neuritis be confused?

With subacute rheumatism, with neuralgia and with disease of bone. The pain occurring in the course of diseases of the spinal cord may be mistaken for an evidence of neuritis.

How is the diagnosis of neuritis to be made?

The symptoms are limited to the distribution of one or more nerves and there is tenderness on pressure. When neuralgia is not dependent on neuritis, there are certain tender points, the pain shoots and intermits, and loss of sensation does not result. Pain of spinal origin is not local in distribution or associated with tenderness.

Multiple Neuritis.

What are the causes of multiple neuritis?

Multiple or *disseminated neuritis*, or *polyneuritis*, may be a complication or a sequel of infectious diseases, such as syphilis, diphtheria and influenza; it may develop in the course of posterior spinal sclerosis or it may appear as a part of anesthetic leprosy; it may be due to lead-poisoning; it may occur endemically, as in the disease known as *kak-kê* or *beri-beri;* the most common causes, however, are chronic alcoholism and exposure to wet and cold.

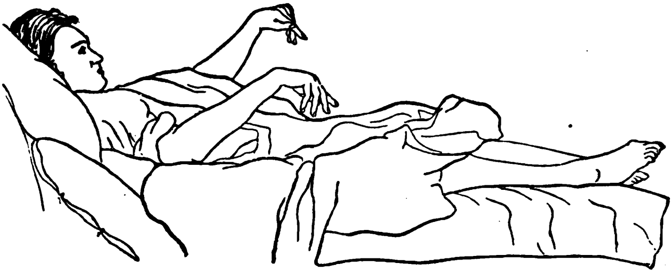
What are the symptoms of multiple neuritis?

If multiple neuritis set in suddenly, it may present rigor, elevation of temperature and other febrile symptoms.

It is attended with numbness, tingling, pain, tenderness and loss of power in the distribution of the nerves affected, usually those of the extremities. Paralysis, wasting and impairment of sensation soon follow. There may be wrist-drop, foot-drop, abolition of reflexes and ataxia.

Trophic changes occur in the skin, nails and hair. Edema is not uncommon. The wasted muscles present reactions of degeneration. Secondary contractions may take place in the unopposed muscles.

FIG. 43.



Multiple neuritis, wrist-drop and foot drop. (Gowers)

Involvement of spinal nerves, such as the phrenic, the vagus or the third, gives rise to symptoms referable to the distribution of those nerves.

How are spinal pachymeningitis and multiple neuritis to be differentiated?

Spinal pachymeningitis is usually cervical ; the symptoms are thus especially referable to the upper extremities. There is also an absence of tenderness in the course of the nerves, while pain radiating from the spinal column is the rule.

How does acute myelitis differ from multiple neuritis?

Myelitis usually involves a limited area of the spinal cord in the dorsal or lumbar region. The symptoms are thus manifested in the lower half of the body and constitute the type of paraplegia. The action of the sphincters is impaired. There is anesthesia, with a zone of hyperesthesia and but little pain. The paralyzed muscles waste rapidly. The reflexes are mostly exaggerated in the parts supplied by the nerves that arise below the affected area of the cord.

Sciatica.

What are the causes of sciatica?

Sciatica is usually dependent upon inflammation of the sciatic nerve.

It is more common in males than in females and in middle life than at any other period. It may be primary, developing in gouty or rheumatic persons or after exposure to wet and cold, or following injury, or as the result of pressure from sitting on the edge of a chair; or secondary in consequence of compression by tumors in the course of the nerve, within or without the pelvis, or as a result of extension from adjacent disease, as of the hip-joint.

What are the symptoms of sciatica?

Sciatica is attended with pain and tenderness in the course and distribution of the sciatic nerve, increased by movement. There are certain tender points: 1, at the posterior inferior spine of the ilium; 2, at the sciatic notch; 3, at the middle of the thigh; 4, on the posterior aspect of the knee; 5, below the head of the fibula; 6, behind the external malleolus; and 7, on the dorsum of the foot. There are also paresthesia, tingling, formication and numbness.

In aggravated cases, the affected muscles waste and present degenerative reactions. There may also be fibrillary contractions and muscular cramps.

Primary sciatica is rarely bilateral.

With what conditions may sciatica be confounded?

With sciatic neuralgia, with disease of the hip-joint, and with disease of the spinal cord or cauda equina.

How is the differential diagnosis to be made?

Sciatic neuralgia occurs in debilitated and anemic persons, with neuralgia elsewhere; the pain is intermittent, and in the distribution rather than in the course of the nerve; it may be induced by movement, but is not otherwise aggravated thereby; there is spontaneous pain rather than tenderness.

Disease of the hip-joint may give rise to pain about the hip-joint and knee; but there is no tenderness in the course of the nerve; and investigation will disclose the obvious cause of the symptoms.

The pain to which disease of the spinal cord or of the cauda equina gives rise is bilateral in distribution; it is not attended with tenderness in the course of the nerve; but it is associated with additional symptoms indicative of its origin.

How are primary and secondary sciatica to be differentiated?

The pain and tenderness in the course of the nerve in primary sciatica are wanting when the sciatica is secondary; the pain is then rather in the distribution of the nerve; other symptoms, and careful examination per rectum, if need be, may lead to the detection of an adequate cause for the pain.

Paralysis of the Facial Nerve.

What are the causes of paralysis of the facial nerve?

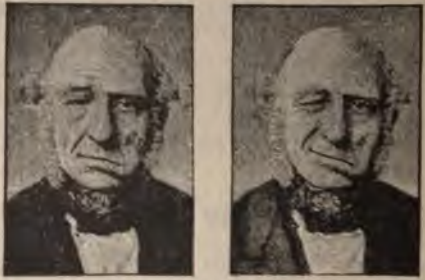
The facial nerve may be paralyzed by lesions above its nucleus (supra-nuclear), by lesions of the nucleus (nuclear), or by lesions in the course of the nerve (infra-nuclear). The first is represented by the paralysis of hemiplegia. The nucleus and the root-fibers may be damaged as a result of hemorrhage, softening, degeneration, or inflammation, or by a new-growth. The nerve may suffer from compression by tumors, from traumatism, from neuritis (primary or secondary). The most common cause of facial paralysis is exposure to cold.

What are the symptoms of paralysis of the facial nerve?

In facial paralysis of nuclear or infra-nuclear origin all of the muscles of one side of the face are paralyzed; the face is rendered asymmetrical by the unantagonized action of the muscles of the opposite side; the eye upon one side remains open and cannot be closed; the lower lid is relaxed and epiphora results; the forehead on that side is smooth and cannot be wrinkled; the angle of the mouth droops; saliva dribbles from the corner of the mouth; the normal furrows are obliterated; the lips cannot be puckered, as in whistling, nor elevated, as in displaying the teeth; associated and emotional movements are wanting on the paralyzed side. Numbness and tingling indicate involvement of filaments of the fifth nerve.

The affected muscles waste and reactions of degeneration develop. If complete recovery do not occur, secondary contraction in the paralyzed and wasted muscles takes place.

FIG. 44.



Facial paralysis. The figure on the right represents an attempt to close the eyes. (Gowers.)

If the seventh nerve is injured between the geniculate ganglion and the origin of the chorda tympani, the sense of taste is lost in the anterior portion of the tongue.

How are facial palsy of supra-nuclear origin and that of nuclear or infra-nuclear origin to be differentiated?

Facial palsy of supra-nuclear origin is but part of a hemiplegia, of which other manifestations will be evident; unlike what takes place in nuclear and infra-nuclear disease, the orbicularis palpebrarum and the occipito-frontal muscles escape. In nuclear and infra-nuclear palsy, muscular wasting takes place, with degenerative reaction, which is not the case in palsy of supra-nuclear origin. In supra-nuclear paralysis, associated and emotional movements of the face are symmetrically performed; in nuclear or infra-nuclear palsy, they are not.

Paralysis of the Phrenic Nerve.

What are the causes of paralysis of the phrenic nerve?

The function of the phrenic nerve may be interfered with by disease of the spinal cord or of the vertebræ, by the pressure of a tumor or of an aneurism, as a result of a neuritis or of a deep wound of the neck.

To what symptoms does paralysis of the phrenic nerve give rise?

When the phrenic nerve is paralyzed, the movements of the diaphragm are enfeebled or abolished. If the nerve on one side is affected, the loss of power is not marked; but if both are paralyzed, the diaphragm does not actively descend in inspiration; instead of the protusion of the upper portion of the abdominal wall, there is retraction; dyspnea is induced by exertion; cough becomes difficult.

From what conditions is paralysis of the phrenic nerves to be differentiated?

From superior intercostal breathing, from inflammation of the diaphragm and from degeneration of the diaphragm. In the first condition, inspiratory contraction of the diaphragm must be carefully looked for; in the second, there has been an adjacent inflammation, most probably of the pleura or peritoneum; the third has only been recognized after death.

If the paralysis of the phrenic nerves is dependent upon disease of the cord, there are present other symptoms than inactivity of the diaphragm.

Paralysis of the Musculo-spiral Nerve.

What are the causes of paralysis of the musculo-spiral nerve?

The musculo-spiral nerve may be paralyzed as a result of traumatism, as from a blow or when the humerus is fractured; the nerve may be compressed by callus, by contraction of the triceps muscle, or by the head of a crutch in the axilla; neuritis may result from exposure to cold. The most common cause of musculo-spiral paralysis is pressure by the head on the arm during sleep.

How is paralysis of the musculo-spiral nerve to be recognized?

The characteristic symptom of paralysis of the musculo-spiral nerve is loss of power in the extensors of the elbow and wrist, in the long extensors of the fingers and thumb, and in the supinators. There is wrist-drop and the fingers and thumb cannot be extended. From the want of antagonism the power of flexion is also enfeebled. Subsequently, the palsied muscles may present reactions of degeneration.

How does the neuritis of lead-poisoning differ from the neuritis of the musculo-spiral nerve resulting from compression or traumatism?

Both are attended by wrist-drop, which in lead-poisoning is bilateral, in compression-neuritis unilateral. In the former, the onset is slow and gradual and the action of the supinators is unimpaired; in the latter, the onset is acute and supination cannot be performed. A blue line on the gums attends lead-poisoning; careful chemic examination during the administration of potassium iodide may detect lead in the urine; while in musculo-spiral palsy a cause of a different kind is readily ascertainable.

Neuromata.

To what symptoms do neuromata give rise?

Tumors in the course of nerves may be composed of nervous structure or be heterologous. They may be hereditary, congenital, or a result of traumatism; they are not uncommonly seen on the stumps of amputated members. The symptoms that such growths occasion necessarily depend upon their situation. At first they give rise to irritation and heightened function, as evidenced by pain and spasm; subsequently they cause abolition of function and paralysis.

Upon what does the diagnosis of neuromata depend?

Upon the detection of a tumor and the obstinacy of symptoms referable to the distribution of one or more nerves.

Neuralgia.

What are the clinical features of neuralgia?

Pain in the course of a nerve, independent of neuritis, appears most commonly in adult life, in anemic and ill-nourished women of emotional temperament. In some instances, an hereditary predisposition can be traced. Among the causes of neuralgia are exposure to cold, rheumatism, gout, malaria, alcoholism, plumbism, traumatism and peripheral irritation.

The pain occurs in paroxysms, in the intervals between which some sensitiveness persists. In the course of the nerve are certain tender points—*points douloureux* of Valleix. Muscular spasm sometimes takes place in the distribution of the motor nerve corresponding to the sensory nerve involved. The paroxysm is sometimes attended with vomiting. Vaso-motor derangement and trophic changes may manifest themselves in the course of the disease. Neuralgia may be localized to a single nerve; it may progress in a radiating manner, or it may change its seat from nerve to nerve.

How are neuritis and neuralgia to be differentiated?

The pain of neuralgia is intermittent and paroxysmal; that of neuritis is continuous; in neuritis there is, in addition, tenderness in the course of the nerve, with swelling. Neuritis is not limited to sensory nerves; in consequence, muscular weakness and, later, wasting and alterations in the electrical reactions are manifest.

What are the symptoms of trigeminal neuralgia?

The symptoms of *neuralgia of the fifth nerve*, *trigeminal* or *trifacial neuralgia*, or *tic douloureux*, depend upon its distribution. One or several branches of the nerve may be involved.

Of the first division, the supra-orbital branch is most commonly affected. Pain is felt on the forehead, on the eyelid, in the eye and on the side of the nose. There are supra-orbital, palpebral, nasal and ocular tender points.

Of the second division of the fifth nerve, the infra-orbital branch is most commonly attacked by neuralgia. Pain is referred to the cheek and ala nasi, between the orbit and the mouth. There are infra-orbital, nasal, malar and gingival tender points.

The pain of neuralgia of the inferior maxillary nerve is referred to the parietal eminence, the temple, the lower jaw, the ear and the tongue. There are inferior dental, temporal and parietal tender points. Sometimes the inferior dental and the auriculo-temporal branches are alone involved.

Migraine.

What are the clinical features of migraine?

Migraine, *hemicrania* or *sick headache* is a paroxysmal neurosis characterized by unilateral headache, associated with nausea, vomiting and derangement of vision and sensation. It is more common in females than in males and in the first half of life than at any other time. Frequently a neurotic heredity can be traced, some members of the same family presenting migraine, epilepsy, neuralgia, insanity or some other neurosis.

The paroxysm often begins with some sensory disturbance, such as tingling or numbness; or with a perversion of vision, such as the appearance of a luminous or brightly colored object; or with an impairment of vision usually presenting the characters of hemianopia or with auditory manifestations, such as tinnitus or a sudden explosive sound. When the headache reaches a considerable degree of intensity nausea and vomiting occur. In extreme cases motor weakness and aphasia are present. The headache, at the beginning of the disease unilateral, may subsequently become bilateral. It is often periodic, and not rarely in some fixed relation with menstruation, which it may accompany, or precede or follow at a certain interval.

It may sometimes be traced to reflex influences, such as may arise from gastric disturbance or eye-strain. These, however, are to be regarded merely as excitants, acting upon a predisposed nervous system, and would not alone be effective.

How are migraine and ordinary headache to be differentiated?

Headache may arise from a multiplicity of conditions—among others from neuralgia, from rheumatism, from gastro-intestinal derangement, from toxemia, from eye-strain, from anemia, from hyperemia and from organic disease of the brain. It is unassociated with other subjective or with visual manifestations; it is bilateral; it is not paroxysmal; it is dependent upon other influences than is migraine.

Spinal Meningitis.

What are the varieties of spinal meningitis?

Spinal meningitis may be *acute* or *chronic;* it may involve the dura, arachnoid or pia, giving rise respectively to *pachymeningitis*, *arachnitis* or *leptomeningitis;* it may be *simple*, *purulent*, *tuberculous* or *hemorrhagic*.

What are the causes of spinal meningitis?

Inflammation of the spinal meninges may result from an extension of adjacent inflammation, from traumatism, from exposure to cold and wet; it may develop in the course of infectious diseases, or as a manifestation of syphilis, of tuberculosis, of pyemia or of alcoholism.

What are the symptoms of acute spinal meningitis?

Meningitis may set in abruptly with a chill, followed by elevation of temperature, pain in the back and radiating in the course of the nerves, hyperesthesia, stiffness and spasmodic contraction of various muscles, exaggerated reflexes, retention of urine and constipation. The pain is intensified in paroxysms. The muscular spasms are induced by efforts at movement. The finger drawn over the skin leaves a red streak.

With the subsidence of the acute symptoms, the pain may yet remain, but the manifestations of irritation give way to those of paralysis: muscular weakness and wasting, anesthesia, abolition of reflexes and enfeeblement of the sphincters. The extent and distribution of the symptoms depend upon the seat of the meningeal inflammation.

How are rheumatism of the muscles of the back and spinal meningitis to be differentiated?

Pain may accompany dorsal or lumbar rheumatism, but it is not radiating, nor is it attended with muscular rigidity, or followed by anesthesia, loss of power and wasting.

Cervical Pachymeningitis.

What are the symptoms of cervical pachymeningitis?

Inflammation of the dura mater in the cervical region is recognized by pain and stiffness in that region, with shooting pains in the arms, and numbness, and impaired sensibility, and loss of power, and wasting. The over-extension of the hand, with flexion of the fingers, that results from the paralysis of the long flexors of the wrist and fingers, and of the interossei gives rise to characteristic deformity. There may also be weakness of the lower extremities.

How is cervical pachymeningitis to be distinguished from progressive muscular atrophy and subacute anterior poliomyelitis?

All three may give rise to wasting and weakness in the upper extremities, and weakness in the lower; but in pachymeningitis sensation is deranged, the wasting is irregular in distribution, and there have at some time been stiffness in the muscles of the neck, and pains in the back and radiating down the arms; while spasm and sensory symptoms are wanting in progressive muscular atrophy and poliomyelitis.

Hemorrhage into the Spinal Membranes.

What are the varieties of hemorrhage into the spinal meninges?

Hemorrhage may take place between the dura and the vertebræ, being then *extrameningeal;* or within the dura, being then *intrameningeal.* Intrameningeal hemorrhage may take place between the dura and the arachnoid, being then *subdural;* or between the arachnoid and the pia, being then *subarachnoid.*

What are the causes of hemorrhage into the spinal meninges?

Hemorrhage into the membranes of the cord is usually a result of traumatism, as from falls or blows, or of violent muscular activity, as during convulsions or labor; it may occur in the course of acute infectious diseases or as a manifestation of a hemorrhagic diathesis; or an aneurism that has eroded the vertebræ may rupture and empty its contents into the spinal canal.

What are the symptoms to which hemorrhage into the spinal membranes give rise?

Hemorrhage into the spinal meninges is marked by sudden, severe pain in the back, to which are soon added rigidity, muscular spasm, radiating pains, hyperesthesia, retention of urine and constipation; should death not occur, these symptoms in turn give way to muscular weakness, anesthesia and derangement of the sphincters.

How are spinal meningitis and hemorrhage into the spinal meninges to be differentiated?

The onset of hemorrhage is sudden, that of meningitis gradual. Meningitis is from the outset attended with elevation of temperature; in hemorrhage, the temperature rises only when meningitis sets in.

Anemia of the Spinal Cord.

What are the manifestations of anemia of the spinal cord?

Anemia of the spinal cord may be part of a general anemia. There is no definite symptom by which it can be recognized. Whatever the symptoms to which it may give rise, they are comprised in those of the condition of which the spinal anemia is a part. There is aching in the legs and the patient fatigues readily. There may be wasting of the extremities and paraplegia.

Hyperemia of the Spinal Cord.

What are the symptoms of hyperemia of the spinal cord?

The clinical recognition of hyperemia, other than as a condition antecedent to inflammation of the spinal cord, is as doubtful as is that of spinal anemia. A diagnosis of hyperemia is justifiable when symptoms apparently indicative of a beginning myelitis disappear in the course of a few days.

Myelitis.

What are the varieties of myelitis?

Myelitis may be *acute*, *subacute* or *chronic;* it may be *transverse*, *focal*, *disseminated*, *diffuse* or *central;* or it may involve only the anterior horns of the gray matter.

What are the causes of myelitis?

Myelitis is more common in males than in females. It may result from traumatism, from exposure to cold and wet, from compression, by extension from adjacent disease and in the course of acute infectious diseases; syphilis may be a cause of myelitis.

What are the symptoms of acute myelitis?

The symptoms of acute myelitis *set in* with variable abruptness and with febrile manifestations. There may at first be pains of moderate severity in the back. Soon, there is loss of motion and sensation in the parts supplied by the nerves arising from the cord below the level of the inflammation, with a girdle-sensation and a zone of hyperesthesia corresponding to the distribution of the nerves given off at the upper limit of the inflammation. Grave trophic changes, as wasting and the formation of bed-sores, may take place, with the development of the reactions of degeneration. The reflexes within the distribution of the nerves arising from the inflamed area are lost; those below are exaggerated. Control of the sphincters is lost.

After the subsidence of the acute symptoms, some improvement slowly takes place, greater in respect to sensation than in respect to motion. Cystitis and consecutive pyelonephritis may develop. Contractures may take place. The inflammation of the cord may extend.

How are acute spinal meningitis and acute myelitis to be distinguished from one another?

The two are likely to be associated; the symptoms of the one or the other, however, predominating. Pain, hyperesthesia and muscular spasm, followed by anesthesia and palsy, characterize acute meningitis. In myelitis, pain and hyperesthesia are slight, transient and circumscribed; muscular spasm is wanting; and wasting, palsy, anesthesia and derangement of the sphincters appear early.

How are acute myelitis and spinal hemorrhage to be differentiated?

The onset of hemorrhage is more abrupt than that of myelitis. Initial fever indicates myelitis rather than hemorrhage. Hemorrhage into the spinal cord is soon followed by myelitis, and the differentiation then becomes impossible.

Chronic Myelitis

What are the causes of chronic myelitis?

Chronic myelitis is more common in males than in females, and in early and middle adult life than at any other period. The same causes that occasion acute myelitis may also give rise to *chronic myelitis.* Thus chronic myelitis may follow syphilis, repeated exposure to cold and wet, repeated over-exertion, compression of the cord, chronic alcoholism and lead-poisoning. Chronic myelitis may be a sequel of acute myelitis or of chronic meningitis.

What are the symptoms of chronic myelitis?

The symptoms of chronic myelitis vary in distribution with the localization of the disease. There is at first an undue readiness of muscular fatigue, soon progressing to weakness and followed by actual palsy. *Sensation* is deranged; paresthesiæ are common; girdle-pain may appear. The *reflexes* are exaggerated. A tendency to spasm develops. The *muscles* waste and present quantitatively altered electric reactions, which, in the later stages of the disease, suffer qualitative change. The *sphincters* usually participate in the loss of motor power.

How does chronic differ from acute myelitis?

The symptoms of chronic myelitis, unlike those of acute myelitis, are slow and gradual in onset, progressive in course, irregular in distribution and unattended with febrile reaction.

What are the distinctions between chronic myelitis and progressive muscular atrophy?

The symptoms of chronic myelitis are irregular in distribution; the manifestations of progressive muscular atrophy appear somewhat symmetrically. Sensory symptoms constitute a distinctive feature of chronic myelitis, but are inconsiderable or wanting in progressive muscular atrophy. Exaggeration of reflexes and spasm characterize myelitis rather than muscular atrophy. Fibrillary muscular contractions and heightened mechanical irritability mark progressive muscular atrophy.

In what respects does chronic myelitis differ from lateral sclerosis?

In lateral sclerosis, sensation is not deranged; there is no girdle-sensation; and the action of the sphincters remains unimpaired.

What is the distinction between chronic myelitis and spinal pachymeningitis?

Spinal pachymeningitis is most commonly cervical and the symptoms are limited to the upper extremities; myelitis irregularly involves all four extremities. Pain is a more constant manifestation of pachymeningitis than of myelitis.

Acute Anterior Poliomyelitis.

What are the causes of acute anterior poliomyelitis?

Acute inflammation of the anterior horns of the gray matter of the spinal cord is more common in males than in females, in late infancy than at any other period of life, and in summer than in winter. It follows sudden chilling of the heated body; injuries to the spine; acute infectious diseases; over-exertion.

What are the symptoms of acute anterior poliomyelitis?

Acute anterior poliomyelitis may set in abruptly, with convulsions, delirium, headache, dull, heavy pains in the back and in the extremities, and febrile symptoms. Soon, extensive palsy appears, followed, after a variable period, by wasting of certain groups of muscles. There may have been incontinence of urine and of feces.

In the course of several weeks the original extent of paralysis gradually diminishes, until ultimately the loss of power and the wasting are limited to certain parts that remain permanently deficient in nutrition, growth and function.

Degenerative reactions of nerve and muscle develop and the reflexes are enfeebled or lost.

What are the distinctions between myelitis and poliomyelitis?

The permanent palsy is more extensive in myelitis than in poliomyelitis. In the former the reflexes are exaggerated; in the latter, enfeebled or lost. Sensory symptoms characterize myelitis; not poliomyelitis. Bedsores are common in the former; not in the latter.

How are the paralysis of poliomyelitis and that of cerebral origin to be differentiated?

The paralysis of poliomyelitis may be limited to one side; but it is not characteristically hemiplegic. It is attended with wasting and the reaction of degeneration; cerebral paralysis may be attended with wasting, but not with the reaction of degeneration. Athetoid movements appear in the course of palsy of cerebral origin; not in the course of the palsy of poliomyelitis. The reflexes are exaggerated in cerebral disease; enfeebled or lost in poliomyelitis.

How are multiple neuritis and anterior poliomyelitis to be differentiated?

Sensory symptoms—tingling, pain, tenderness, numbness and anesthesia—as seen in neuritis, are wanting in poliomyelitis. Multiple neuritis is rather symmetrical, poliomyelitis irregular in distribution. The one is the more common in adults; the other, in children.

Acute Ascending Paralysis.

What are the symptoms of acute ascending paralysis or Landry's paralysis?

The nature of acute ascending paralysis is not yet known. The disease is thought to depend upon a toxic influence exerted upon the nervous system. It arises under conditions similar to those that precede acute myelitis. It is characterized by progressive motor paralysis, beginning in the lower extremities and gradually extending upwards. The power of movement is lost; respiration, deglutition, and articulation are interfered with. Numbness and tingling are sometimes present at the onset and followed by impairment of sensation. The reflexes are enfeebled. Trophic changes are wanting. The sphincters are usually involved. The spleen is enlarged. Most cases terminate fatally.

How are acute myelitis and acute ascending paralysis to be differentiated?

Febrile symptoms, anesthesia, trophic changes and involvement of the sphincters do not attend acute ascending paralysis (Landry's disease). Recovery from acute ascending paralysis is less common than is recovery from myelitis.

How is acute ascending paralysis to be distinguished from multiple neuritis?

The view has been expressed that acute ascending paralysis is dependent upon multiple neuritis. Acute ascending paralysis, however, does not appear in all four extremities at once, and is said not to present the alterations of sensation, the muscular wasting and the elevation of temperature of multiple neuritis.

What is the distinction between acute ascending paralysis and anterior poliomyelitis?

The onset of acute anterior poliomyelitis is, while that of acute ascending paralysis is not, attended with febrile manifestations. In poliomyelitis, the paralyzed muscles waste; in ascending paralysis, they do not. Poliomyelitis is rarely fatal; ascending paralysis is usually fatal.

Amyotrophic Lateral Sclerosis — Progressive Muscular Atrophy—Glosso-Labio-Laryngeal Palsy.

There are three affections intimately related to one another: *amyotrophic lateral sclerosis*, *progressive muscular atrophy* and *glosso-labio-laryngeal palsy.* The pathologic lesions are similar in all: a degenerative process in the gray matter and in the conducting paths. The symptoms of the three affections differ according to the situation of the disease in the gray matter, and according to whether the change begins first in the gray matter and then involves the white or *vice versa.*

If the white matter of the lateral columns is first involved, with partial or with subsequent invasion of the gray matter of the anterior horns, amyotrophic lateral sclerosis results. If the gray matter of the cord be first involved, the symptoms of progressive muscular atrophy appear; while if the process extend to or originate in the gray matter of the medulla the symptoms are those of bulbar palsy, superadded or occurring isolated.

What are the causes of amyotrophic lateral sclerosis, progressive muscular atrophy and chronic bulbar palsy?

In many cases, no cause can be determined; in others the symptoms have been preceded by mental strain, by exposure to cold and wet, by concussion, by syphilis, by lead-poisoning, and by acute diseases; in some there is an indirect neurotic heredity. All three affections are more common in males than in females, and in middle life than at any other period.

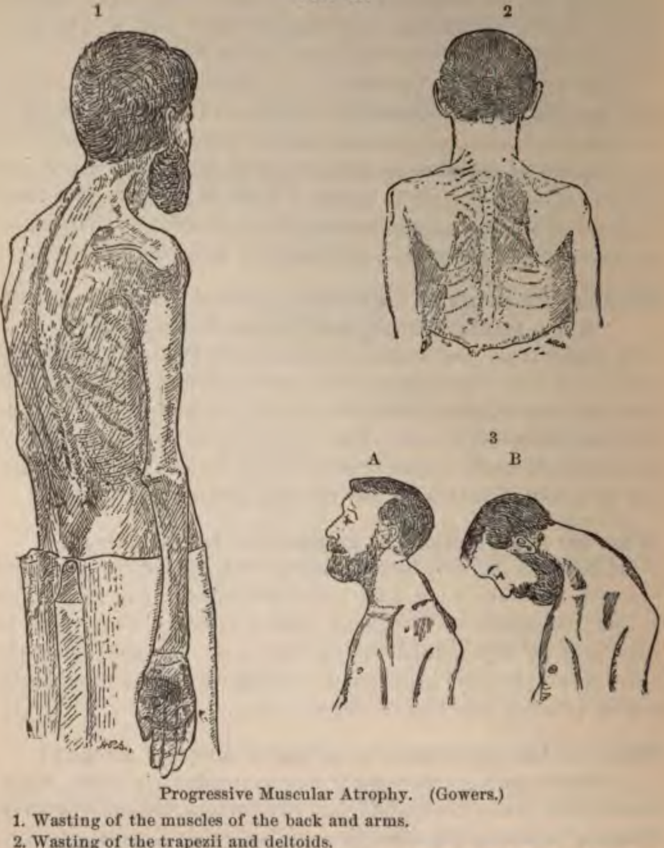
What are the symptoms of amyotrophic lateral sclerosis?

Amyotrophic lateral sclerosis begins with symptoms of lateral sclerosis: weakness; a stiff, awkward, spastic gait; muscular spasm; exaggerated reflexes; spinal epilepsy; to which in turn may be superadded the symptoms of degeneration of the anterior horns of the gray matter: muscular wasting, paralysis, loss of reflexes, reaction of degeneration.

What are the symptoms of progressive muscular atrophy?

Progressive muscular atrophy is usually insidious in onset; sometimes slow, sometimes rapid, but always progressive in course. Muscular wasting, preceded by pain, is first observed in one portion of the body, usually in an upper extremity, and followed by weakness; both wasting and weakness in turn successively invade all four extremities and the trunk as well; respiration may be embarrassed. If the duration of the case be long enough, symptoms of bulbar paralysis are superadded.

Fig. 45.



Progressive Muscular Atrophy. (Gowers.)

1. Wasting of the muscles of the back and arms.
2. Wasting of the trapezii and deltoids.
3. Wasting of the muscles of the neck; A, habitual posture of the head; B, position into which the head falls if it be not inclined backward.

The wasting is especially manifest in the thenar and hypothenar eminences and in the interosseous spaces; as a result, a peculiar deformity—the claw-like hand—develops. The wasting muscles are the seat of spontaneous fibrillary contractions. Mechanical muscular irritability is heightened. The deep reflexes are enfeebled in a degree proportional to the muscular wasting. The electric reactions are also dependent upon the nutrition of the muscles and nerves. At first, they present quantitative, subsequently qualitative changes.

What are the symptoms of chronic or progressive bulbar paralysis—glosso-labio-laryngeal palsy?

The symptoms of *glosso-labio-laryngeal paralysis* are analogous to those of progressive muscular atrophy, the degenerative process, however, involving the nuclei of the cerebral rather than those of the spinal nerves, especially the facial, glosso-pharyngeal, pneumogastric, spinal accessory and hypoglossal. The disease is marked by difficulty in mastication, in deglutition, in respiration, in phonation and articulation, and by wasting of the muscles concerned in these functions. The affected muscles display spontaneous fibrillary contractions. Speech becomes progressively more difficult and indistinct and finally nasal. Swallowing is difficult and fluids regurgitate through the nose. Saliva dribbles from the mouth. Food may find its way into the larynx. Reflex action in the throat may be lost. Rarely the ocular and the facial muscles participate in the palsy.

How is progressive muscular atrophy to be distinguished from acute anterior poliomyelitis?

Progressive muscular atrophy is especially a disease of adult life. It is usually gradual in onset and progressive in course, while acute anterior poliomyelitis is especially a disease of childhood, and is acute in onset and retrogressive in course. Febrile symptoms attend acute poliomyelitis, but not progressive atrophy. The fibrillary twitching of the latter is not present in the former. Terminal bulbar symptoms attend progressive muscular atrophy, but not acute poliomyelitis.

Acute Bulbar Palsy.

What is acute bulbar palsy?

Symptoms almost identical with those of progressive glosso-labio-laryngeal paralysis may set in acutely. They may subsequently undergo some improvement and then remain stationary. The condition is dependent upon an acute lesion—softening or hemorrhage or inflammation of the medulla, involving the nuclei of the glosso-pharyngeal, pneumogastric, spinal accessory and hypoglossal nerves or the fibers passing to or from them.

How is acute to be distinguished from chronic bulbar palsy?

The symptoms of chronic bulbar palsy are gradual in onset, progressive in course and symmetrical in distribution; those of acute bulbar palsy are sudden in onset, regressive or stationary and are apt to display slight irregularities of distribution.

Pseudo-Bulbar Palsy.

What is pseudo-bulbar palsy?

A bilateral lesion, such as softening or hemorrhage, involving the lower portion of the ascending frontal convolution of both hemispheres, or the corresponding motor tracts in symmetrical situations, will give rise to the symptoms of bulbar palsy: paralysis of the lips, tongue and throat.

How is pseudo-bulbar palsy to be distinguished from true bulbar palsy?

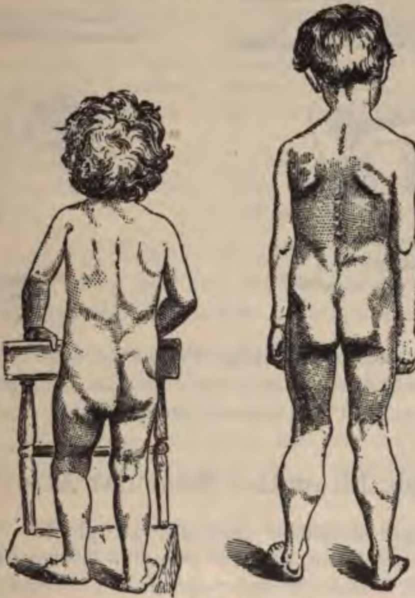
Pseudo-bulbar palsy, in contradistinction to true bulbar palsy, is likely to be characterized by two distinct attacks, by slight asymmetry of distribution of the symptoms, and by the absence of wasting.

Pseudo-Hypertrophic Paralysis.

What are the symptoms of pseudo-hypertrophic paralysis?

Pseudo-hypertrophic paralysis is a congenital family-affection, transmitted through females but occurring more commonly in males, usually appearing in childhood, and characterized by weakness and enlarged muscles, the fibers of which undergo atrophy, while the interstitial tissue increases. The muscles of the calves and the infra-spinati usually participate in the enlargement in striking degree. Wasting of the latissimus dorsi and of the lower portion of the pectoralis major is characteristic.

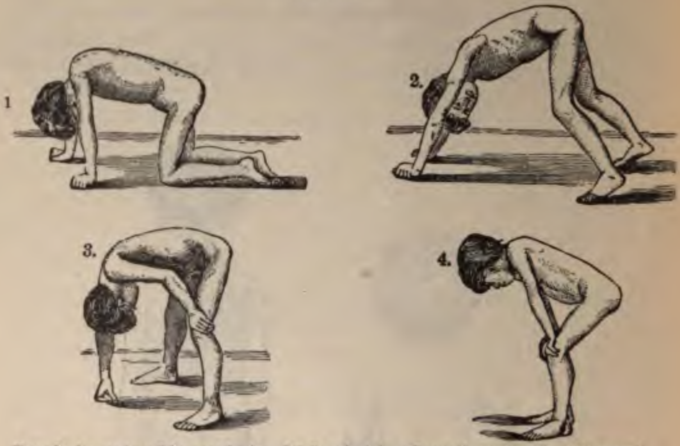
FIG. 46.



Pseudo-hypertrophic paralysis in two brothers. (Gowers.)

The gait has a peculiar oscillating character; ascending stairs is difficult, and the manner of rising from the floor is characteristic (Fig. 47): the patient first gets on his hands and knees; then extending the legs he stands upon his feet; finally, by supporting his hands upon his thighs he manages to reach the erect posture. Ultimately the muscles become reduced in size; the electric reactions suffer quantitative changes, indica-

FIG. 47.



Pseudo-hypertrophic paralysis. Mode of rising from the ground. (Gowers.)

tive of diminished excitability; the deep reflexes are enfeebled and lost. Talipes equinus, spinal curvature and muscular contractions occur at a late stage of the disease.

Simple Idiopathic Muscular Atrophy.

What is simple idiopathic muscular atrophy?

There is a condition, allied to pseudo-hypertrophic paralysis, in which the muscles atrophy, but do not become enlarged by interstitial deposit. The affection occurs in families, is as common in females as in males, and usually appears in early adult life.

What are the symptoms of simple idiopathic muscular atrophy?

Simple idiopathic muscular atrophy sets in gradually, with weakness and wasting in the face, arms or legs and extension to the rest of the body. The deep reflexes become enfeebled, deformities result and the electric reactions are quantitatively changed. Among other muscles, the latissimus dorsi, the infraspinati, the zygomatici and the orbicularis oris show a special proneness to undergo wasting and palsy.

What are the distinctions between pseudo-hypertrophic muscular paralysis and simple idiopathic muscular atrophy?

PSEUDO-HYPERTROPHIC MUSCULAR PARALYSIS.	SIMPLE IDIOPATHIC MUSCULAR PARALYSIS.
More common in males.	Equally common in the two sexes.
Some muscles enlarged; gastrocnemii and infraspinati especially prone to enlargement.	Muscles usually wasted, especially the orbicularis and the zygomatici.
Face not involved as a rule.	Involvement of face characteristic.
Notable increase of interstitial tissue in muscles.	Slight or no increase of interstitial tissue in muscles.

Other cases, typical of one or of the other form of idiopathic muscular atrophy, will be found among the members of the family of either, respectively.

What are the distinctions between progressive muscular atrophy and simple idiopathic muscular atrophy?

Simple idiopathic muscular atrophy is a disease that occurs in families, appearing in childhood, the muscles of the face, especially the orbicularis oris and the zygomatici, being involved.

Progressive muscular atrophy is a disease independent of family influences, appearing in adult life, the wasting often beginning in the hands, while the face remains uninvolved.

Arthritic Muscular Atrophy.

What is arthritic muscular atrophy

It has been observed that in the course of inflammation of a joint, acute or chronic, spontaneous or traumatic, the related muscles, especially the extensors, undergo a varying degree of atrophy. With the disappearance of the arthritis, the affected muscles gradually return to their normal condition. If the joint-disorder continue for a long period, the deep reflexes in the region involved are heightened and the muscles present quantitative electrical changes; contracture of the opponent muscles may also occur.

Thomsen's Disease—Myotonia Congenita.

What are the symptoms of myotonia congenita—Thomsen's disease?

The disorder described by Thomsen is one that occurs as a congenital affection in families. It is characterized by tonic muscular spasm on voluntary movement following a period of rest. If movement is persevered in, the spasm relaxes. The spasm is intensified by emotion or by the fear of its occurrence. Muscular hypertrophy is the ultimate result. A peculiar electric reaction, *myotonia electrica*, is developed.

Posterior Spinal Sclerosis—Locomotor Ataxia.

What are the causes of posterior spinal sclerosis?

Posterior spinal sclerosis, *locomotor ataxia*, or *tabes dorsalis* is more common in males than in females, and in middle adult life than at any other period. Its most common cause is syphilis; other causes are exposure to cold and wet, concussion, overexertion and sexual and alcoholic excesses; the disease has been observed to follow acute infectious diseases; it is sometimes secondary to other forms of spinal disease; in some cases an hereditary neurotic influence can be traced. It has been attributed to metallic poisoning. The etiology is sometimes obscure.

What are the symptoms of posterior spinal sclerosis?

Posterior spinal sclerosis is characterized by impairment of coördination, giving rise to difficulty in performing delicate movements, to unsteadiness of gait and of station, particularly when the guidance of vision is removed; by shooting pains in the extremities; by a girdle-sensation; by other abnormalities of sensation, particularly anesthesia, and especially in the soles of the feet; by abolition of the deep reflexes; by primary exaggeration and secondary loss of the superficial reflexes; by primary increase of the sexual propensity and secondary impairment of the sexual power; by derangement of the sphincters, manifested by retention or incontinence; by narrow pupils that act in accommodation, but do not respond to the stimulus of light; by paralysis of ocular muscles, giving rise to strabismus and diplopia; by atrophy of the optic and auditory nerves, occasioning loss of vision and of hearing; by laryngeal, gastric, intestinal and other visceral crises, manifested by paroxysms of intense distress; by trophic changes in the joints, giving rise to enlargement and subluxation, rendering the bones brittle and liable to spontaneous fracture; by perforating ulcers of the foot; and in the last stages by muscular wasting.

How is multiple neuritis to be distinguished from posterior spinal sclerosis?

If abolition of the reflexes, unsteady gait and station, loss of sensation and sharp pains attend neuritis, they may recede and ultimately disappear; once present in the course of posterior spinal sclerosis they persist. Posterior sclerosis is progressive in course, and unyielding in treatment. With appropriate treatment neuritis is retrogressive. The girdle-sensation of sclerosis is wanting in neuritis. In neuritis, the muscles undergo degeneration and waste; there is palsy; the electric reactions are qualitatively changed. In sclerosis, muscular wasting and weakness result only after long-continued inactivity; if the electric reactions undergo any change, it is quantitative. In neuritis there are pains and points of special tenderness in the course and in the peripheral distribution of various nerves; in posterior sclerosis the muscles are not tender.

How are lumbar pachymeningitis and posterior spinal sclerosis to be differentiated?

Pachymeningitis in the lumbar region may be attended with shooting pains in the thighs and with abolition of the knee-jerks, but not with manifestations indicative of involvement of cerebral nerves. In meningitis the muscles waste early; in posterior sclerosis, not at all or only late. Impairment of co-ordination characterizes posterior sclerosis.

What are the distinctions between posterior spinal sclerosis and cerebellar tumor?

A tumor in or compressing the middle lobe of the cerebellum may give rise to incoördination, to a staggering gait and to abolition of the knee-jerks; it gives rise in addition to occipital headache, to vertigo, vomiting, nystagmus, optic neuritis and to other evidences of compression; while lightning-pains and alteration of sensation are wanting.

Primary Lateral Sclerosis—Spastic Paraplegia.

What are the causes of primary lateral sclerosis?

Primary lateral sclerosis or *spastic paraplegia* occurs with equal frequency in both sexes, and a little earlier in life than posterior sclerosis.

In many cases, no etiologic element can be discovered; in others, there is a history of concussion, of exposure to cold and wet, of syphilis, of excesses or of neurotic heredity.

What are the symptoms of primary lateral sclerosis?

Primary lateral sclerosis, or spastic paraplegia, is characterized by muscular weakness and spasm; the latter usually involves the extensors of the lower extremities occasioning a peculiar spastic gait and so-called "clasp-knife" rigidity; attacks of "spinal epilepsy" occur; the reflexes are exaggerated; ankle-clonus is present. There is usually muscular wasting, occasionally hypertrophy. The arms are affected less commonly and in less degree than the legs. There may be abnormal sensations, but no anesthesia. The sphincters may be involved.

The electric reactions undergo quantitative changes.

How is lateral sclerosis to be distinguished from myelitis?

While lateral sclerosis presents the symptoms of degeneration of the lateral columns, as seen in myelitis, it is always gradual in onset and unattended with febrile manifestations, with girdle-pain, or with impairment of sensation. Typical paraplegia, with muscular wasting and degeneration and impairment of the sphincters is less characteristic of lateral sclerosis than of myelitis.

How may lateral sclerosis simulate and be distinguished from cerebral hemiplegia?

The manifestations of primary lateral sclerosis may be unilateral; but the face is not involved, as it usually is in hemiplegia of cerebral origin; and careful examination will detect exaggeration of the reflexes of the upper as well as of the lower extremity on the apparently uninvolved side, unlike what is found in cerebral hemiplegia.

Postero-lateral Sclerosis—Ataxic Paraplegia.

What are the causes of ataxic paraplegia or postero-lateral sclerosis?

Postero-lateral sclerosis, or *ataxic paraplegia*, is more common in males than in females and in middle adult life than at any other period. Its etiology is often obscure. In some cases a neurotic heredity can be traced; in others there has been exposure to cold and wet; in still others concussion seems to have been the cause; a history of syphilis is uncommon.

What are the symptoms of postero-lateral sclerosis, or ataxic paraplegia?

The symptoms of ataxic paraplegia are dependent upon sclerosis of the posterior and lateral columns of the spinal cord. The disease is gradual in onset and marked by muscular weakness, spasm and incoördination. There may be pain in the sacral region, but lightning-pains are absent. Articulation may be defective. Tremor of the muscles of the face may occur. The deep reflexes are exaggerated. The iris usually reacts to light. Atrophy of the optic nerve is rare. Sexual power may be lost. The sphincters become enfeebled. Ultimately, contractures develop.

How is posterior spinal sclerosis to be distinguished from postero-lateral sclerosis?

POSTERIOR SCLEROSIS.	POSTERO-LATERAL SCLEROSIS.
Syphilis a common cause.	Syphilis a rare cause.
Weakness a late symptom.	Weakness an early symptom.
Knee-jerk lost early.	Knee-jerk exaggerated.

POSTERIOR SCLEROSIS.	POSTERO-LATERAL SCLEROSIS.
No ankle-clonus.	Ankle-clonus.
Never muscular spasm.	Characteristic muscular spasm.
Argyll-Robertson pupil.	Pupil responds to light.
Lightning-pains.	Dull sacral pains.
Sensory impairment.	Sensation unimpaired.
Girdle-sensation.	No girdle-sensation.
Optic-nerve atrophy common.	Optic-nerve atrophy rare.
Visceral crises.	No crises.

How is postero-lateral sclerosis to be distinguished from primary lateral sclerosis?

Ataxic paraplegia is to be distinguished from spastic paraplegia by the presence of symptoms of muscular incoördination, dependent upon involvement of the posterior columns of the cord.

What are the distinctions between a tumor of the cerebellum and postero-lateral sclerosis?

A growth involving the middle lobe of the cerebellum may give rise to weakness, ataxia, spasm, and heightened reflexes; there are, besides, occipital headache, optic neuritis, vertigo, vomiting and nystagmus; other evidences of pressure may be present.

Friedreich's Ataxia—Hereditary Ataxic Paraplegia.

What are the symptoms of Friedreich's ataxia?

The symptoms of *Friedreich's ataxia* are dependent upon sclerosis of the lateral and posterior columns of the spinal cord. The disease occurs in families and appears early in life; it attacks both sexes alike. It is attended with an ataxic gait, impairment of coördination and equilibration, muscular weakness, abolition of knee-jerks, nystagmus and defective speech; sensation is usually unimpaired.

What are the distinctions between Friedreich's ataxia and ataxic paraplegia?

The early period of life at which the symptoms appear, its occurrence in families, the absence of knee-jerks and of ankle-clonus and the presence of nystagmus distinguish Friedreich's disease from ataxic paraplegia.

Cerebro-Spinal Sclerosis—Insular Sclerosis.

What are the causes of cerebro-spinal sclerosis?

The more commonly-recognized causes of *disseminated*, *multiple*, *insular*, or *cerebro-spinal sclerosis* are exposure to cold and wet, traumatism, nervous shock and acute febrile diseases. Occasionally a neuropathic heredity can be traced.

What are the symptoms of cerebro-spinal sclerosis?

The symptoms of disseminated, multiple; insular, or cerebro-spinal sclerosis vary with the distribution of the islets of sclerosis. As a rule, there is a peculiar jerky incoördination or tremor, most marked in the upper extremities, and sometimes involving the head and the tongue, aggravated by effort, emotion or observation ; there is commonly nystagmus, usually lateral, sometimes vertical, sometimes rotatory ; speech is often slow, scanning, syllabic, parts of words being dropped ; the reflexes are usually exaggerated. In addition there are certain mental changes, often manifested by a sense of complacency, contentment and self-satisfaction, quite at variance with the patient's condition. Evidences of muscular weakness are not rarely present. There may also be headache, vertigo and optic neuritis.

How is cerebro-spinal sclerosis to be distinguished from postero-lateral sclerosis?

There may sometimes be considerable difficulty in differentiating cerebro-spinal sclerosis and postero-lateral sclerosis (ataxic paraplegia) and Friederich's (hereditary) ataxia. In postero-lateral sclerosis, the defect of coördination involves the lower extremities primarily ; in multiple sclerosis, there is coarse tremor rather than true incoördination, and the upper extremities are especially involved. Nystagmus occurs in Friedreich's ataxia and in multiple sclerosis, but not in ataxic paraplegia. The knee-jerks are exaggerated in multiple sclerosis and in ataxic paraplegia, but are wanting in Friedreich's ataxia. Friedreich's ataxia is a family-disease and appears early in life. Multiple sclerosis and ataxic paraplegia appear in middle life. Cerebral symptoms and mental phenomena belong to multiple sclerosis rather than to postero-lateral sclerosis. Speech is slow and scanning in cerebro-spinal sclerosis; in Friedreich's ataxia there are merely elision and occasional separation of syllables.

Paralysis Agitans—Shaking Palsy.

What conditions favor the development of paralysis agitans?

Paralysis agitans or *shaking palsy* begins most commonly at about fifty years of age. It is more common in males than in females. In some cases, an hereditary influence can be traced in an etiologic connection. In others, the onset of the disease has been preceded by decided emotional disturbance, by physical injury or by acute disease.

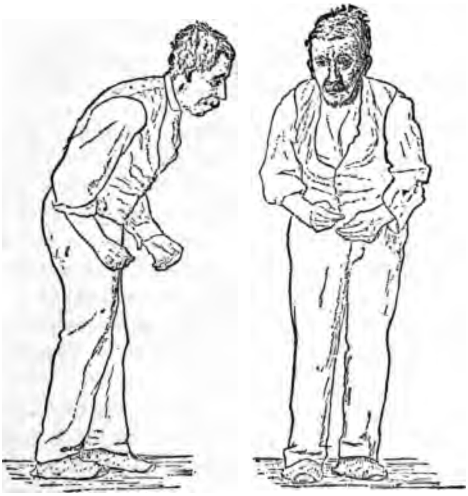
What are the symptoms of paralysis agitans?

Paralysis agitans, shaking palsy or Parkinson's disease is characterized by tremor, weakness and rigidity. Tremor is usually first observed; weakness and rigidity subsequently. The manifestations first appear in one extremity, and gradually extend to the others. The tremor is fine and rhythmical; in its incipiency it may be absent during rest and induced by movement: in its characteristic form, it is constant during the waking hours. As a rule, the head does not participate in the tremor; exceptionally it does. The expression of the face is fixed and immobile. The shoulders are bent forward, giving rise to the phenomena of propulsion—a tendency to run forward. Occasionally there is retropulsion, or there may be a tendency to lateral movement. The hands assume a peculiar, semi-flexed attitude, the fingers performing movements as if rolling a small object between their tips; or the fingers are held as if grasping a pen. The voice is monotonous; words are uttered rapidly, with a tendency to confluence of syllables. The reflexes are usually unaltered; exceptionally the knee-jerks are exaggerated and an ankle-clonus can be elicited. A sensation of abnormal heat, sometimes with perspiration, is often present.

What are the differential features between paralysis agitans and cerebro-spinal sclerosis?

The diagnosis between these two conditions, ordinarily simple, may under some circumstances be extremely difficult. The tremor of cerebro-spinal sclerosis is coarse and irregular, and is induced and aggravated by voluntary effort, by emotion and by observation; that of paralysis agitans is fine and regular, and is constant, at least during the waking hours. In cerebro-spinal sclerosis, the head participates in the movements; in paralysis agitans, on the contrary, the face is fixed, immobile, expressionless. Paralysis agitans does not present the slow, scanning speech and nystagmus of multiple sclerosis; while the latter does not present the tendency to forward or backward or even lateral movement of the former. The characteristic attitude of the hand, as if holding a pen in writing, or as if rolling pills between the fingers, seen in paralysis agitans, is not seen in multiple sclerosis.

FIG. 48.



Paralysis agitans. (After St. Leger.)

Spinal Hemorrhage.

What are the causes of hemorrhage into the spinal cord?

Spinal hemorrhage is more common in males than in females, and in adult life than at any other period. It may be *primary*, dependent upon disease of the bloodvessels, or as a result of exposure to cold, or of over-exertion, or of sexual excess; *secondary*, in the course of inflammation, tumors and cavities in the cord; *accessory*, occurring towards the close of convulsive disorders; and *traumatic* following blows, falls and other injuries.

What are the symptoms of hemorrhage into the spinal cord?

The occurrence of hemorrhage into the spinal cord is indicated by sudden, severe pain in the back, with loss of motion and sensation. Consciousness may be lost, but is likely to be preserved. A girdle-sensation exists at the level of the lesion, and loss of motion and sensation below, on alternate sides, or on both sides of the body, according to the seat of the lesion. The sphincters are likely to be deranged and trophic changes to occur. Respiration will be interfered with if the lesion is in the cervical or dorsal region of the cord.

Some degree of myelitis and meningitis are usually developed in the progress of the case, giving rise to fever and irritative symptoms. Subsequently, the symptoms become paralytic in type. Some degree of paraplegia remains permanently. The paralyzed muscles waste. The deep reflexes are exaggerated.

What are the distinctions between spinal hemorrhage and spinal meningitis?

The abruptness of onset is more decided in hemorrhage than in meningitis. Febrile manifestations attend meningitis from the beginning; they only appear in hemorrhage when myelitis is established. Motor and sensory impairment is more decided in hemorrhage than in meningitis; in the latter, there is a preliminary stage of spasm and pain.

How are hemorrhage into the cord and hemorrhage into the meninges to be distinguished from one another?

Both the local and the radiating pains are less severe in spinal than in meningeal hemorrhage, while the subsequent anesthesia is more decided in the former than in the latter. In the case of spinal hemorrhage the paralytic symptoms are more decided from the outset; while in meningeal hemorrhage these are preceded by muscular spasm. Trophic changes characterize spinal hemorrhage, and are wanting in meningeal hemorrhage.

Spinal Compression.

What are the causes of compression of the spinal cord?

The cord may be compressed by tumors of the spinal canal; by dislocation of the vertebræ, independently, or as a result of caries, or of fracture; by an exostosis; by an aneurism that has eroded the bones; or by an hydatid cyst.

How can the causes of spinal compression be differentiated?

The recognition of a tumor in the spinal canal depends upon its detection from without, or the detection of new-growths in other parts of the body.

Simple dislocation and fracture of the vertebræ follow traumatism; the symptoms to which dislocation gives rise set in suddenly and are profound in degree; a deformity of the spinal column may be detectable.

The diagnosis of caries depends upon the knowledge of a history of syphilis or of tuberculosis and the detection of a painful deformity in the back.

An hydatid cyst of the spinal canal may be diagnosticated by the detection externally of a fluctuating tumor, upon puncture of which the characteristic hooklets may be found.

It may be impossible to diagnosticate the cause of compression of the cord dependent upon an exostosis or upon an aneurism.

To what symptoms does compression of the spinal cord give rise?

The rapidity with which the symptoms of compression appear depends somewhat upon the cause. Dislocation is apt to occasion manifestations of immediate gravity. In other cases, the symptoms are gradual in appearance and progressive in course.

Compression gives rise to two groups of phenomena, referable to the nerve-roots and to the cord, respectively. There is local pain in the back, aggravated by movement, as well as pains of a radiating character, with girdle-sensation; ultimately anesthesia develops. There is loss of motion in the parts supplied by the nerves from the cord below the seat of compression; with exaggerated reflexes and involvement of the sphincters. The palsied muscles slowly waste and degenerative reactions set in. Contractures may develop.

How is compression-myelitis to be distinguished from hemorrhage into the spinal cord?

When a vertebra softened by destructive disease suddenly gives way, the resulting displacement may be followed by compression of the cord, occasioning symptoms with which those produced by hemorrhage into the cord may be identical. The symptoms of compression, however, are usually more extensive and more absolute than those of hemorrhage; the existence of a deformity of the spine makes the diagnosis certain.

What are the distinctions between compression of the cord and chronic myelitis?

Symptoms of irritation referable to the nerve-roots are wanting in myelitis. The recognition of a cause of compression determines the diagnosis.

Tumor of the Spinal Cord.

What are the symptoms of tumor of the spinal cord?

Tumors of the spinal canal may be situated without or within the dura mater, or within the structure of the cord itself. They are most diverse in character. The symptoms will depend upon the situation of the tumor and upon the degree of mechanical interference with the functions of the cord that it occasions. Pressure on the nerve-roots gives rise to pain, to abnormal sensations, to girdle-pain and to muscular spasm and rigidity. Pressure on the cord or myelitis occasions anesthesia, paralysis and exaggerated reflexes below the level of the lesion and abolition of the reflexes within the area innervated from the seat of the growth. Involvement of the anterior horns of the gray matter is attended with wasting and other trophic disorders ; involvement of the lumbar enlargement causes abolition of the knee-jerk, loss of control of the sphincters and wasting and palsy in the lower extremities ; involvement of the cervical enlargement occasions wasting and palsy in the upper extremities. The unilateral appearance of symptoms of spinal disease, or evidence of sensory derangement on one side and motor derangement on the other, is strongly suggestive of tumor of the spinal canal. With the growth of the tumor the symptoms become bilateral.

From what conditions is spinal tumor to be distinguished?

The diagnosis of spinal tumor includes the determination of the nature of the tumor and its distinction from other conditions presenting similar symptoms.

In the first connection, a history of syphilis, of tuberculosis, or of tumors situated elsewhere is to be considered.

The differentiation from caries of the vertebræ depends upon the recognition of disease of the bone, upon the deformity that results and upon the greater degree of pain on movement that attends caries.

From hypertrophic pachymeningitis spinal tumor differs in course ; being progressive, while symptoms of irritation precede those of paralysis and wasting. The symptoms of pachymeningitis are more circumscribed in distribution than are those of tumor.

The symptoms of tumor differ from those of myelitis, in being irritative in character—attended by pain and spasm, rather than paralytic. If tumor give rise to myelitis, the recognition of the condition depends upon a knowledge of the previous symptoms.

The persistence of obstinate neuralgia, especially if bilateral, should excite suspicion of spinal tumor.

Syringomyelia.

What are the symptoms of syringomyelia?

Cavities in the spinal cord may be a result of defective apposition of the lateral halves of the cord in the course of development; of occlusion of the central canal, with accumulative distention by cerebro-spinal fluid; of the disintegration of gliomata; or they may appear subsequently to myelitis. The course of the disease is slow and chronic, sometimes covering many years. The symptoms vary somewhat with the situation of the cavity. They are usually most conspicuous in the upper extremities. There are muscular wasting and weakness, preceded by alterations of sensation. Common sensibility is usually preserved, while the perception of pain and of heat and cold is enfeebled or lost. In some cases, severe pains occur. A spastic condition may be present in the lower extremities. The sphincters are often involved. Trophic changes are common. There may be cutaneous eruptions, as eczema or herpes. The skin may be thin and glossy or thick and horny. Vaso-motor disturbance may be manifested by coldness and lividity.

From what conditions is syringomyelia to be differentiated?

Myelitis, hypertrophic pachymeningitis and progressive muscular atrophy occasion certain symptoms in common with syringomyelia.

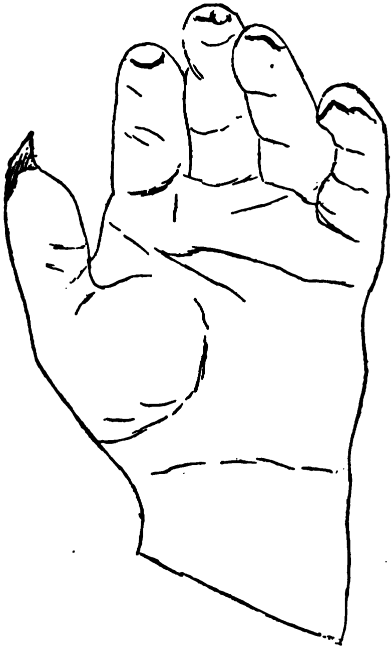
Myelitis is recognized by the much more profound palsy and loss of sensory power, without involvement of the pain-sense and the temperature-sense; hypertrophic pachymeningitis by the attendant pain and the less extensive anesthesia; and chronic muscular atrophy by the absence of conspicuous sensory symptoms.

Morvan's Disease—Analgesic Panaris.

What are the symptoms of Morvan's disease?

Under the name of *Morvan's disease* has been described a syndrome of symptoms, including the development of a painless inflammation at the extremities of the fingers, followed by necrotic sequestration of the phalanges. At the beginning of the disease, the affected parts may be the seat of pain. Subsequently, analgesia develops together with the destructive process in the fingers. In most cases abnormal curvature of the spinal column has been observed. After death, hyperplasia of the connective tissue of the peripheral nerves and in the posterior horns, posterior columns, and the gray matter of the cervical segment of the spinal cord has been found.

FIG. 49.



Appearance of the hand in Morvan's Disease. (After Charcot.)

What is the distinction between Morvan's disease and scleroderma?

Scleroderma is a morbid condition in which, as a result of inflammatory changes in the subcutaneous arteries of a varying distribution, hyperplasia of the connective tissue takes place, with hardness, swelling and fulness, which in turn is succeeded by contraction and atrophy. The condition may be circumscribed or diffuse. It is sometimes attended with pigmentation and sometimes with desquamation. The etiology of the affection is obscure.

A condition in which the extremities of the fingers close to nails undergo discoloration, followed by the formation of bullæ, with perhaps loss of the nails and shortening of the fingers, has been described as a variety of scleroderma or *sclerodactylia.* When nose and ears and face, as well as hands and feet, become reduced in size, the name *ukromikria* has been employed. In scleroderma there may be anesthesia, but there is not analgesia. Trophic changes may take place in the affected parts, but they are not of a necrotic character. The nails may be lost, but there is no exfoliation of bone.

How are Morvan's disease and anesthetic leprosy to be differentiated?

In anesthetic leprosy, a destructive process may be set up in the extremities, attended sometimes with the loss of fingers or toes, but the condition is usually to be recognized by the association of other definite symptoms of leprosy, such as patches of anesthesia and leprous nodules in various parts of the body.

From what other conditions is Morvan's disease to be differentiated?

Syringomyelia may present trophic changes and analgesia; but there is usually also loss of temperature-sense; while necrosis of bone is wanting and common sensibility is preserved.

Raynaud's disease is attended with cyanosis of the extremities, sometimes terminating in gangrene; but sensibility remains unimpaired; and there are other characteristic symptoms.

Syphilis may occasion a destructive dactylitis. The diagnosis will depend upon the absence of sensory symptoms and upon a knowledge or a history of infection or of other manifestations.

Necrosis of bone may attend *diabetes*, but sensory symptoms are wanting, distinctive symptoms are present and examination of the urine will make clear the clinical association.

Cerebral Meningitis.

What are the varieties of inflammation of the membranes of the brain?

Inflammation of the dura, pia or arachnoid is known as *pachymeningitis*, *leptomeningitis* or *arachnitis*, respectively; most commonly the pia and arachnoid are involved together—*pia-arachnitis* or *leptomeningitis.* Inflammation of the dura mater may be attended with the extravasation of a membranous hemorrhagic exudate—*hemorrhagic pachymeningitis.* Meningitis may especially involve the *convexity* or the *base;* it may be *acute* or *chronic*, *primary* or *secondary;* it may be *simple*, *purulent*, *tuberculous*, *syphilitic.*

What are the causes of cerebral meningitis?

Inflammation of the pia and arachnoid may be a result of traumatism or of contiguous inflammation; it may develop in the course of infectious disease or of pyemia; it may depend upon a deposit of tubercles in the membranes.

What are the symptoms of cerebral meningitis?

The symptoms of meningitis vary somewhat, as the base or the convexity of the brain is involved. Tuberculous meningitis is usually basilar. Meningitis dependent upon causes other than tubercles, usually, though not exclusively, affects the convexity. Certain symptoms are common to meningitis in any situation. In addition to those of the associated condition, there are, together with febrile manifestations, headache, delirium, vomiting, convulsions, retraction of the head, derangement of the sphincters, palsies, vaso-motor and trophic disturbances, inequality of the pupils and irregularity of the pulse and respiration. When the base is involved, there are optic neuritis followed by atrophy, irritation followed by paralysis of cranial nerves and more decided alteration of pulse and respiration, trophic and vaso-motor disturbances and retraction of the head. When the inflammation is tuberculous, tubercles may, on ophthalmoscopic examination, be detected in the choroid.

What are the symptoms of cerebral pachymeningitis?

Inflammation of the dura mater may be a result of traumatism or of adjacent disease. In addition to the symptoms of the primary condition, there may be headache, delirium, convulsions, febrile manifestations. A collection of pus may occasion paralysis.

What are the symptoms of hemorrhagic pachymeningitis?

Hemorrhagic pachymeningitis is more common in males than in females, and late in life than at any other period. It is usually preceded by a history of alcoholism, of insolation, of traumatism, or of insanity; it may also develop in the course of blood-diseases. It is attended with headache, contracted pupils, intellectual torpor, apoplectiform convulsions, unnatural drowsiness and mental wandering. The condition is rather obscure, and the symptoms not well defined. It is often unexpectedly found *post mortem.*

How is simple cerebral meningitis to be distinguished from tuberculous meningitis?

Ordinary cerebral meningitis sets in rather acutely; tuberculous meningitis usually more insidiously. The ordinary form of inflammation is associated with well-known causes: traumatism, adjacent inflammation, infectious diseases and pyemia; tuberculous meningitis is usually secondary to tuberculous processes at other parts of the body. The symptoms of the former especially indicate involvement of the convexity and freedom of the base, while in the latter the conditions are reversed, and choked disc and paralysis of cranial muscles are more common. Tubercles are never found in the choroid in ordinary meningitis. Simple meningitis is not uncommonly attended with recovery; tuberculous meningitis but rarely.

How are the cerebral symptoms of acute general disease to be distinguished from the symptoms of cerebral meningitis?

Cerebral symptoms may appear at any time in the course of acute general diseases, such as scarlatina, smallpox, yellow fever, typhoid fever, rheumatism, pneumonia and acute miliary tuberculosis. Investigation should be directed to the determination of the association, so that the primary condition may not be obscured. Sufficient cause for the symptoms may be found in the high temperature, in the state of inanition, or in the poisoned condition of the blood. When the delirium of febrile affections appears, the headache that has been present disappears. Headache and delirium continue together in meningitis. The manifestations of irritation and paralysis of cranial nerves, including optic neuritis and atrophy, seen in meningitis, are wanting in simple febrile disorders.

How is cerebral meningitis to be distinguished from cerebro-spinal fever?

The differences are of degree, extent and etiology. When the membranes of the brain are inflamed, the spinal membranes are usually in some degree also involved. Cerebro-spinal fever is considered a constitutional affection, cerebral meningitis a local affection. In the former, however, the spinal involvement stands boldly out, as evidenced by the tendency to opisthotonos, the symptoms on the part of the trunk, the sphincters, and the lower extremities; while the latter is especially marked by the involvement of cerebral structures. Cutaneous eruptions mark cerebro-spinal fever, but are no part of cerebral meningitis. The recognition of a cause of cerebral meningitis, or a knowledge of the existence of other cases of cerebro-spinal fever may determine the diagnosis.

How is cerebral meningitis to be distinguished from acute mania?

The mental wandering of meningitis does not usually attain the high degree seen in mania; and in the former it is attended with febrile manifestations, in the latter not. The symptoms on the part of the cranial nerves, the headache, the convulsions of meningitis are wanting in mania.

Hydroc

What are the varieties of hydr

An excessive accumulation
place in the subdural space
brain. The one condition is ca
hydrocephalus. Either may be
ondary, congenital or *acquired.*

What are the causes of hydroce

Hydrocephalus may be a resu
of the orifices of communicatio
between the fourth ventricle and
pressure on the veins of Gale
factor is recognizable.

To what symptoms does hydrocep

Occurring in a child prior to t
the complete ossification of the
separation and a thinning of th
fluid distends the ventricles, the
becomes attenuated. Under suc
larged ; the fontanels may be open
tal condition is defective ; there i
may be convulsions ; and various
lyzed.

In the more aggravated cases idio
with choked discs ; and life may b
and coma.

Analogous symptoms also occur wi
later in life.

Hemorrhage into the Cer

What are the varieties of hemorrhage

The extravasation of blood without
the dura and the bone, constitutes
hemorrhage within the dura mater, bet

noid is called *subdural;* and between the arachnoid and pia mater, *subarachnoid.*

Under what conditions does hemorrhage into the meninges of the brain occur?

Hemorrhage into the cerebral membranes may be a result of traumatism; of the rupture of an aneurism of a meningeal vessel; of the rupture of a hemorrhage in the brain; of the same conditions as occasion cerebral hemorrhage; it may occur in the insane; rarely it occurs spontaneously.

To what symptoms does hemorrhage into the cerebral membranes give rise?

The symptoms of hemorrhage into the cerebral membranes, depend upon the volume and the extent of the extravasation. The symptoms are those of meningitis, plus those of cerebral hemorrhage, and may be preceded by headache, vertigo and vomiting.

The infantile meningeal hemorrhages that result during labor occasion the so-called birth-palsies, with the symptoms of congenital spastic paraplegia or hemiplegia.

Congenital Spastic Paraplegia.

What is infantile or congenital spastic paraplegia?

When, as a result of injury to the child during birth, bilateral meningeal hemorrhage takes place over the central convolutions, the pyramidal tracts fail to develop or they degenerate. The child is late in learning to walk. It presents the symptoms of spastic paraplegia. The gait is peculiar, one foot being placed over or in front of the other, or a swinging oscillation taking place. Growth and development are retarded. Athetoid movements in the hands are common. Initial convulsions may occur and be repeated, subsequently to cease. Should the hemorrhage be circumscribed or unilateral, the distribution of the symptoms will vary accordingly. There is usually defective mental development.

What are the distinctions between congenital spastic paraplegia and pseudo-hypertrophic paralysis?

The reflexes are exaggerated in congenital spastic paraplegia, enfeebled or lost in pseudo-hypertrophic paralysis. The gait of congenital spastic paraplegia is spastic; that of pseudo-hypertrophic paralysis is oscillating; the manner in which the child rises from the floor in the latter is characteristic. In congenital spastic paraplegia the contractures are yielding; in pseudo-hypertrophic paralysis unyielding; the former is retrogressive, the latter progressive. Should there be decided enlargement of some of the muscles, this will constitute a diagnostic feature.

Cerebral Anemia.

What are the conditions that lead to cerebral anemia?

Anemia of the brain may be general: as a part of a systemic anemia, from cardiac insufficiency, from accumulation of blood elsewhere, from pressure on the large vessels to the head, from pressure on the brain; or partial: from obstruction of the circulation by vascular occlusion or by pressure from without. Cerebral anemia may develop gradually or suddenly.

To what symptoms does cerebral anemia give rise?

General anemia of the brain, suddenly induced, occasions the symptoms of syncope: failure of vision, ringing in the ears, vertigo, nausea, shallow, sighing respiration, contracted pupils, pallor of the face, a cool, moist skin and loss of consciousness. Nystagmus and convulsions may occur. The loss of consciousness may pass into coma, and coma into death.

General anemia of the brain, gradually induced, occasions enfeeblement with irritability of function. There are headache, vertigo, impaired intellection, motor weakness, drowsiness or insomnia, hallucinations, mania or melancholia. The optic disc may be pale. The symptoms are aggravated by the erect posture and may be mitigated by recumbency or inhalation of amyl nitrite.

Partial anemia of the brain is followed by impaired nutrition and loss of function in the affected area. Occurring suddenly, it is attended with loss of consciousness and convulsions; when of gradual development, it occasions headache, vertigo, numbness, tingling and weakness.

Cerebral Hyperemia.

What conditions lead to cerebral hyperemia?

Hyperemia of the brain may be active or passive. *Active hyperemia* may be caused by an overacting left ventricle; by sudden contraction of the vessels elsewhere; by insolation; it may be part of a general plethoric condition; and it occurs as the first stage of the inflammatory process.

Passive hyperemia is a result of cardiac insufficiency, of venous obstruction as a result of pressure from without or of pulmonary disease.

To what symptoms does cerebral hyperemia give rise?

The symptomatology of cerebral hyperemia is somewhat obscure. The recognition of the condition is not always easy. Usually there are dull headache, a sense of fulness of the head, vertigo, mental torpor, derangement of sleep, a disinclination to activity, flashes of light and tinnitus aurium. The countenance may be flushed; the vessels of the eyeground injected. There may be transient loss of consciousness. Convulsions are uncommon.

Cerebritis.

What are the clinical features of cerebritis?

Inflammation of the structure of the brain may be acute or chronic. It is usually a result of traumatism, or it may arise by extension from adjacent disease. Some degree of cerebritis is coincident with inflammation of the membranes. The symptoms are rather obscure and ill-defined. There are headache, vertigo, delirium, convulsions and febrile symptoms. Cerebritis may be followed by abscess.

How is inflammation of the membranes to be distinguished from inflammation of the structure of the brain?

Inflammation of the membranes and inflammation of the structure of the brain are to some degree always associated, the symptoms of either condition respectively predominating. Inflammation of brain-tissue, however, is usually attended with symptoms more profound and more depressing than are those of meningitis; while the symptoms of meningitis are usually more widely distributed and more irritative in character.

Cerebral Abscess.

What are the causes of abscess of the brain?

The development of cerebral abscesses is usually dependent upon traumatism, suppurative disease of adjacent structures or pyemia. Distant suppuration may give rise to embolic abscess. Inflammation of the brain may terminate in suppuration and the formation of an abscess. Cerebral abscess may be acute or chronic in its course; it may be single or multiple.

What are the symptoms of abscess of the brain?

The symptoms of *abscess of the brain* present themselves in three stages: a *primary acute stage*, in which the symptoms of the associated condition may obscure the cerebral symptoms; a *secondary stage of lull*, in which the symptoms are latent and in abeyance; and a *terminal stage*, in which rupture of the abscess gives rise to the abrupt appearance of the signs of meningitis, of cerebritis or of apoplexy, followed by stupor, coma and death. These stages are not always distinct. Commonly the one passes by gradations into the other.

The more obvious symptoms are fever, recurrent rigors, headache (which may be localized), vertigo, vomiting and optic neuritis (which are most marked in cerebellar abscess), epileptiform convulsions and paralysis (when the abscess is seated in the motor area). There may be, in addition, unilateral ptosis, defect of speech, impairment of articulation, difficulty of deglutition and mental disturbance.

From what conditions is abscess of the brain to be differentiated?

Abscess of the brain gives rise to many of the symptoms of tumor of the brain. In the terminal stage it occasions the symptoms of meningitis, of cerebritis or of apoplexy. The distinction from these several conditions depends upon the recognition of a cause of abscess, upon the recurrence of chills, upon the presence of febrile symptoms. The headache and optic neuritis of tumor are more intense than those of abscess. In meningitis there is greater involvement of cranial nerves than in abscess. The course of the symptoms of abscess is of longer duration than is the case in cerebritis. Optic neuritis is wanting in apoplexy. The symptoms of the terminal stage of abscess are to be distinguished from meningitis, cerebritis and apoplexy by a knowledge of the previous existence of obscure symptoms of cerebral disease.

Cerebral Hemorrhage.

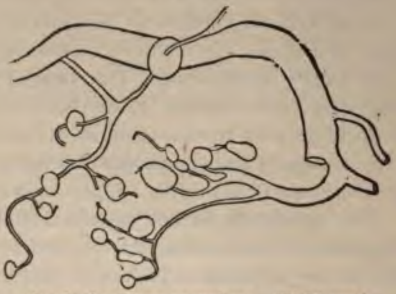
What are the causes of cerebral hemorrhage?

The ultimate cause of cerebral hemorrhage, not traumatic in origin, is disease of the bloodvessels. The event may be induced by over-action of the heart. In some cases of cerebral hemorrhage, an hereditary tendency can be elicited. Hemorrhage in the brain is most common at the degenerative period of life, and its freqency increases with age; the condition occurs more commonly in males than in females; in temperate than in tropical climates; in winter than in summer. The antecedent disease of the vessels may depend upon alcoholism, gout, rheumatism, syphilis, nephritis, lead-poisoning, infectious diseases and blood-diseases.

What is the pathology of cerebral hemorrhage?

The symptoms that follow the occurrence of cerebral hemorrhage depend upon laceration of the brain-tissue, and upon the compression and irritation of adjacent structure to which the extravasation gives rise. In the course of time, the blood, as well as the products of disintegration, are absorbed, until there remains to mark the previous hemorrhage nothing but a fibrous cicatrix or a cyst. The conducting motor fibers related to the structures destroyed undergo secondary degeneration.

FIG. 50.



Cerebral miliary aneurisms. (Eichhorst.)

The most frequent seat of cerebral hemorrhage is the region of the central ganglia; next in frequency is the centrum ovale; then the cortex, pons and cerebellum.

What are the symptoms of cerebral hemorrhage?

The occurrence of hemorrhage in the brain is indicated by sudden, complete loss of consciousness, with general muscular relaxation, and depression of temperature. True prodromata are wanting. The breathing is labored and stertorous; an initial convulsion may occur; the pulse is apt to be slow, full and hard; the pupils unequal; the reflexes abolished or unequal; and the head and eyes deviated to one side (conjugate deviation). Urine and feces may be passed involuntarily. The urine may contain albumin. Vomiting may occur. As the shock of the seizure passes off, consciousness slowly returns and the evidences of hemiplegia become apparent. The leg, the arm, perhaps the face, are paralyzed on the side opposite to that on which the lesion is situated. The tongue, protruded, deviates to the paralyzed side. There is difficulty in deglutition, in phonation, in articulation. There may be partial or complete anesthesia of the paralyzed members. Secondary fever appears. Gradual improvement slowly takes place, in greatest degree in those parts that habitually act in association with corresponding parts of the opposite side.

If the hemorrhage occur in the cortex, epileptiform convulsions take place; if in the posterior part of the third frontal convolution of the left side, aphasia results; if in the posterior part of the first temporal convolution, word-deafness; if in the angular gyrus and occipital lobe, hemianopsia and word-blindness; if in the anterior part of the uncinate convolution, loss of the sense of taste. If the hemorrhage is large and extensive the patient may never emerge from the primary coma, but death may speedily or slowly take place. Implication of the pons or medulla is indicated by abnormal elevation of temperature. In most cases the hemorrhage involves the region of the central ganglia and the internal capsule.

Athetosis, post-hemiplegic chorea and other spasmodic conditions may be sequelæ of cerebral hemorrhage. The reflexes become exaggerated, especially upon the paralyzed side, as a result of descending degeneration of the pyramidal tracts. The memory is impaired. Emotional mobility is increased. Various vasomotor and trophic changes may take place in the affected parts, which may also become fixed by contractures.

In what respects do the symptoms of cerebral hemorrhage differ from those of cerebral congestion?

Cerebral congestion may be attended with transient loss of consciousness, not so profound, however, as is the case in hemorrhage. The event is prone to be repeated, but the condition does not give rise to permanent paralytic manifestations.

What are the distinctions between the loss of consciousness of syncope and that of cerebral hemorrhage?

Syncope is usually a result of cerebral anemia, induced by insufficiency of the circulation; it may also result from nervous influences that stimulate the vagus. The loss of consciousness is of but brief duration. The pulse is feeble, the face pale, the breathing shallow and sighing. The reflexes are preserved. The sphincters are continent. The return to consciouness is not attended with evidences of paralysis. The attack is prone to be repeated.

How is the coma of cerebral hemorrhage to be distinguished from the coma of alcoholic intoxication?

The symptoms of cerebral hemorrhage come on suddenly; those of alcoholic coma, less suddenly, as a direct consequence of the imbibition of a toxic quantity of alcohol. The coma of alcoholism is not so profound as that of hemorrhage. It may be possible by appropriate stimuli to rouse an individual overcome by alcohol, but there is no response from the coma of hemorrhage. The odor of alcohol in the breath, and the detection of alcohol in the urine would be doubtful evidence; but the absence of these conditions would exclude alcoholism. Conjugate deviation of the head and eyes is a common feature of hemorrhage; alcoholism will not cause it. The pupils are usually dilated in alcoholism, not contracted and unequal as in hemorrhage. The temperature-alterations of hemorrhage are wanting in alcoholic intoxication. On the return of consciousness after alcoholic coma, there is no palsy; palsy is characteristic of hemorrhage.

How is the coma of cerebral hemorrhage to be distinguished from that of opium-poisoning?

In the absence of the previous history, cerebral hemorrhage is to be distinguished from opium-poisoning by the primary depression and secondary elevation of temperature, by the conjugate deviation of the head and eyes, by the evidences of paralysis.

The small pupils of opium-poisoning are equal; if hemorrhage causes contraction of the pupils, one is likely to be smaller than the other. The noisy breathing of hemorrhage does not become so infrequent as the breathing in opium-poisoning.

How is uremia to be distinguished from cerebral hemorrhage?

The conditions under which uremia develops predispose to the occurrence of cerebral hemorrhage. The recognition of disease of the kidney does not help in diagnosis. The coma of hemorrhage, however, is likely to set in more suddenly and to be more profound than is that of uremia.

Uremia is more likely than hemorrhage to appear in young persons. The convulsions that attend uremia are general; unilateral convulsions are indicative of cerebral hemorrhage. The primary depression of temperature is followed by a secondary elevation in hemorrhage, but usually not in uremia. Inequality of the pupils and conjugate deviation of the head and eyes are indicative of hemorrhage, rather than of uremia.

If loss of muscular power follow uremia, it is circumscribed and transitory; in hemorrhage, it is usually hemiplegic and permanent.

In uremia the tongue is dry and covered with a thick brownish fur; there is often vomiting; the body exhales a peculiar, musty odor, not observed in hemorrhage.

How is asphyxia to be distinguished from cerebral hemorrhage?

When an individual is asphyxiated from whatever cause, the appearance is cyanotic. Respiration is interfered with, but is not noisy. There is no conjugate deviation of the head and eyes. Measures of resuscitation may, in a little while, prove successful, while the paralytic phenomena attendant upon cerebral hemorrhage are absent.

Cerebral Softening.

What are the varieties of softening of the brain?

Softening of the brain may be acute or chronic. *Acute softening of the brain* may be a result of inflammation; it may follow vascular occlusion, thrombotic or embolic, arterial or venous.

Chronic softening of the brain is an affection of the degenerative period of life. It usually involves the white matter of the hemispheres. The symptoms to which it gives rise are not well defined. There is usually progressive loss of motor power and impairment of sensation, sometimes with rigidity. Mental deterioration may be slight or considerable. Life may be prolonged for from two months to two years, death usually resulting from bedsores, pneumonia, or some other intercurrent malady.

Cerebral Embolism.

Under what conditions does cerebral embolism occur?

A vegetation from a diseased cardiac valve may be washed into one of the vessels of the brain; or the plug may have its source in a thrombus that has formed in one of the cavities of the heart, or in a calcareous fragment from the wall of an atheromatous aorta.

To what symptoms does cerebral embolism give rise?

The sudden plugging of one of the vessels of the brain is usually manifested by transient loss of consciousness of varying duration, often accompanied by convulsions. On the return to consciousness, the symptoms of hemiplegia are evident, often right-sided, and associated with aphasia. In the course of a few days, secondary fever appears. Unilateral convulsions may be repeated. Choreoid, athetoid, and other disorders of movement are likely to develop.

How do the symptoms of cerebral hemorrhage differ from those of cerebral embolism?

Cerebral embolism is a disease rather of early life; hemorrhage is one of advanced life. Hemorrhage occurs in association with disease of the vessels; embolism with valvular disease of the heart or other source of emboli. Consciousness is invariably lost when hemorrhage takes place; it is more likely not to be in cases of embolism. Embolism is more likely to be cortical and to give rise to initial and consecutive epileptiform convulsions, to localised palsies and to aphasia; hemorrhage is more likely to be central, to be unattended with convulsions, and to present hemiplegia. Both direct (local) and indirect (general) symptoms are more profound and more extensive in hemorrhage than in embolism. The detection of emboli in other organs is an important element in diagnosis.

Cerebral Thrombosis.

Under what conditions does cerebral thrombosis occur?

The most common cause of cerebral thrombosis is vascular disease, whether atheromatous, syphilitic or fibroid. The blood may also coagulate in the vessels in conditions of lowered vitality and enfeebled circulation. Increased coagulability of the blood predisposes to the occurrence of thrombosis. A thrombus may form from an embolus by extension.

To what symptoms does cerebral thrombosis give rise?

The actual occurrence of thrombosis in a cerebral vessel may for a long time be preceded by distressing headache, vertigo, numbness, tingling, muscular weakness, impairment of memory and mental failure. The formation of the thrombus is announced by loss of consciousness, of varying degree and duration, perhaps attended with convulsions or with delirium. In the course of a few days, decided secondary fever may appear. Subsequently, the evidences of hemiplegia become manifest; while recurring localized convulsions may take place. As in the case of embolism, involuntary and spasmodic disorders of movement in the affected parts are common. Thrombosis of the sinuses gives rise to localized edema of the face and scalp.

How are cerebral hemorrhage and cerebral thrombosis to be differentiated?

Thrombosis, unlike hemorrhage, is of not rare occurrence in infancy and extreme old age. The symptoms of hemorrhage set in suddenly, with loss of consciousness; those of thrombosis are preceded by prodromata and develop gradually, with headache, delirium, perhaps convulsions. The convulsions of thrombosis are wont to be localized or unilateral; if convulsions attend hemorrhage, they are usually general. Hemorrhage is the more likely to occur under conditions of excitement; thrombosis, amid conditions of depression. In hemorrhage the circulation is active, the arterial tension high; thrombosis is not uncommonly associated with a stagnant circulation in a debilitated individual. The symptoms are more profound and extensive in hemorrhage than in thrombosis. Mobile spasm, athetosis and other disorders of movement more commonly follow thrombosis than hemorrhage. Recurrent convulsions also point to thrombosis rather than to hemorrhage.

Cerebral Tumor.

What are the more common varieties of tumor of the brain?

Cerebral tumors are most commonly gliomata, tuberculomata, sarcomata, gummata, myxomata, carcinomata or psammomata. They may develop in the cerebral mass, in the membranes, or in the bone.

Not rarely, cysticerci or echinococcus cysts develop in the brain. Gliomata sometimes follow injuries to the head.

Tuberculomata appear in young persons, and are usually associated with tuberculosis elsewhere than in brain. Carcinomata occur later in life, give rise to a cachexia, and are associated with similar growths elsewhere. Gummata are present with other manifestations and a history of syphilis.

What are the symptoms of tumor of the brain?

The symptoms vary somewhat with the size and situation of the new-growth. Certain phenomena, however, characterize the presence of a neoplasm in any part of the brain. There is persistent headache, intensely aggravated in paroxysms; optic neuritis followed by atrophy; mental failure; impaired memory; emotional mobility; vomiting; vertigo; sometimes forced movements; slowness of speech; separation of syllables; confluence of articulation; sometimes aphasia; paralysis of varying distribution, associated with or followed by contractions; sometimes choreoid or athetoid movements, or a jerky incoördination; convulsions resembling those of major or of minor epilepsy, or of Jacksonian epilepsy, or attacks of transient convulsions; alterations of sensation; palsy of cranial nerves; sometimes conjugate deviation of the head and eyes; abnormalities of the pulse; interference with the respiration. The quantity of urine evacuated may be increased; there may be albuminuria or glycosuria. Finally, stupor and coma may be the precursors of death.

How may chronic nephritis simulate cerebral tumor; how are the two to be differentiated?

Chronic nephritis may be attended with headache, vertigo, vomiting, optic neuritis and convulsions, but the urine contains casts as well as albumin, edema exists, the arterial tension is heightened, the heart is hypertrophied, and the palsies of tumor are wanting.

What symptoms does anemia have in common with tumor of brain, and how are the two to be differentiated?

Both anemia and cerebral tumor may occasion headache, vertigo, optic neuritis and albuminuria, but the headache of anemia is not so intense as that of tumor; the optic neuritis of anemia is more rapid in development than is that of tumor; while the pallor, the breathlessness, the edema, the submissiveness to judicious treatment of the one, and the convulsions, the palsies, the progressiveness, and the resistance to treatment of the other are distinctive.

How are the epileptiform convulsions of cerebral tumor to be distinguished from the convulsions of essential epilepsy?

In themselves the convulsions may be indistinguishable, though those of tumor are the more likely to be local or unilateral. Epilepsy, however, fails to present the distinctive symptoms of tumor: headache, vertigo, vomiting, optic neuritis and palsies.

How may the symptoms of cerebral tumor be mistaken for those of cerebro-spinal sclerosis; how is the diagnosis to be made?

A tumor in the brain may occasion a peculiar jerky incoördination, but it does not occasion the nystagmus, the peculiarity of speech, the exaggeration of reflexes of cerebro-spinal sclerosis, while headache and optic neuritis are more common and more intense.

Intracranial Aneurism.

To what symptoms does an intracranial aneurism give rise?

Aneurism of the cerebral vessels may develop as a result of vascular disease, syphilitic or otherwise, of traumatism and of embolism.

The symptoms of intracranial aneurism necessarily vary with the situation of the aneurism. The more constant symptoms are those of an intracranial tumor: headache, vertigo, vomiting, convulsions, palsies, optic neuritis. The detection of a bruit or the demonstration of symptoms dependent upon the presence of a tumor in the course of a cerebral vessel would be diagnostic.

The occurrence of rupture is indicated by the symptoms of cerebral hemorrhage.

General Paralysis of the Insane.

What are the symptoms of general paralysis of the insane?

General or *progressive paralysis of the insane*, or *paretic dementia*, is a fatal disease of insidious invasion, occurring in those who have abused their mental and physical energies, and characterized by psychic alteration and failure, with progressive palsy.

It is much more common in men than in women, and in early middle life than at any other period.

Among the first symptoms are an alteration of disposition, and a change of character. An individual, previously more or less refined, affectionate, attentive, scrupulous, methodical, precise and neat, becomes careless, indifferent, negligent, coarse, indecent, irritable and forgetful. He fails to keep appointments. He makes ill-judged investments and extravagant expenditures. He eats irregularly and hastily, and sometimes eats and drinks excessively, and sleeps poorly.

The palsy is not of the ordinary type. There is at first loss of coördinating power for delicate tasks, such as writing or painting, while general muscular strength, as for lifting, is but slightly impaired. The paralysis is associated with tremor. The pupils may be contracted, sluggish and unequal.

To these symptoms are added the so-called delusions of grandeur, which vary from time to time. The patient believes himself fabulously wealthy. He projects colossal enterprises, or announces wonderful discoveries. He is, however, contented with his surroundings. In a milder form, the delusions take the less obtrusive shape of an invincible, unreasoning optimism, and may be overlooked. In other cases there are alternations of depression and exaltation. Sleeplessness becomes more obstinate. Speech becomes defective, indistinct and stuttering. In writing, words, syllables, letters are omitted or misplaced or repeated. There is a peculiar tremor of the lips. The protruded tongue presents both rhythmical tremor and fibrillary contractions. The gait becomes shuffling. Control over the limbs is progressively lost. At this stage, apoplectiform and epileptiform seizures may take place, and paroxysms of rage and fury may occur. Sensibility is not impaired until late, when it may be almost abolished.

Deceptive lulls and remissions may interrupt the progress of the disease, but finally a condition of terminal dementia is reached. Nutrition gradually fails. Paralysis, mental and physical, becomes so great that death may result from the entrance of food into the air-passages. Coma, convulsions, painful contractions, obstinate diarrhea, pulmonary affections, especially pneumonia, are among the phenomena preceding death. At the autopsy chronic congestive, inflammatory and degenerative changes may be found in the brain and membranes and in the posterior and lateral columns of the spinal cord.

How are progressive paralysis of the insane and posterior spinal sclerosis to be differentiated?

As the symptoms of posterior spinal sclerosis may be superadded to those of progressive paralysis of the insane, the differentiation of uncomplicated cases from locomotor ataxia, as from cerebro-spinal sclerosis and paralysis agitans, essentially depends upon a recognition of the presence or absence of those manifestations of central dissolutional processes that characterize progressive paralysis: the changes in character and disposition, the emotional disturbances, the impairment of memory, the maniacal outbursts, the delusions of grandeur, the peculiar tremor of lips and tongue, the inequality of the pupils, the derangement of the language-faculty in speech and writing, the progressive failure of voluntary motion and intellection, the epileptiform and apoplectiform attacks.

Acute development of the peculiar loss of coördinating power, mental and physical, without obvious cause, in a man of middle life, especially one given to mental, emotional, venereal or alcoholic excess, should at once excite a suspicion of the development of paretic dementia.

Sunstroke.

What are the symptoms of sunstroke?

Sunstroke, *heat-stroke*, *insolation*, of which there are at least two principal varieties, *heat-fever* or *thermic fever* and *heat-exhaustion*—is an affection of the heated term. It occurs in persons exposed to intense heat, solar or artificial, in whom the respiratory and cutaneous transpiration is checked.

Crowding and defective ventilation predispose to the occurrence of heat-stroke. The *onset* may be abrupt or gradual, the symptoms severe or mild (incomplete). While most often coming on during active muscular exertion, as in the case of laborers and marching soldiers, the attack not infrequently follows the mid-day meal. It usually but not invariably happens during the period of maximum heat of the day.

There occur increased frequency of micturition, headache, vertigo, nausea, vomiting, delirium, loss of consciousness, dysphagia, stertorous breathing and coma, associated in the febrile form with a hot, often flushed, skin, frequent pulse and high temperature. The pupils may be dilated or contracted, or contraction and dilatation may be present at different times in the same case. There may be rectal and vesical incontinence, sometimes partial suppression of urine. The duration of the symptoms varies from a few minutes to several hours.

Death may occur early or late; *recovery* may be speedy or tardy. Persistent headache, vesical irritability, choreoid movements of the upper extremities, mental impairment, epilepsy, and in rare instances hemiplegia, or other paralysis, usually transient, may supervene as *sequelæ*. Anhydrosis, with headache and elevation of temperature, annually recurring near the anniversary of the attack, has been observed.

General paralysis of the insane (paretic dementia) has been attributed to sunstroke.

What are the principal points of differentiation between heat-exhaustion and heat-fever, or sunstroke proper?

In heat-exhaustion the skin is pale, cool or cold, moist or clammy; the temperature is low, even subnormal; there is syncope rather than coma; the pupils are usually dilated; the pulse is feeble; convulsions are absent; and rapid relief follows the use of warmth and stimulants.

How does cerebral hemorrhage differ from sunstroke?

The coma of sunstroke, unlike that of cerebral hemorrhage, does not set in unannounced; nor is it as lasting or as profound; while dysphagia may be greater, stertor is less.

The symptoms of sunstroke are symmetrical; the pupils and the reflexes are equal; hemiplegia is not an ordinary sequel.

The temperature rises much higher in insolation than at any time in hemorrhage. The pulse is frequent, often feeble; that of cerebral hemorrhage is commonly slow and full.

It must, however, not be forgotten that cerebral hemorrhage may occur amid circumstances favorable for the development of sunstroke.

With what other conditions might sunstroke be confounded?

Sunstroke might be mistaken for acute alcoholism, meningitis, uremia or narcotic poisoning. Alcoholic excess may bring on an attack, so that the phenomena of both might be intermingled. The history and the symptoms detailed should prevent error in other cases.

In uremia, too, convulsions usually precede coma.

Delirium Tremens.

What are the symptoms of delirium tremens?

The continued ingestion of excessive quantities of alcohol sometimes gives rise to a condition in which with impaired appetite and gastric irritability are associated inability to sleep, tremor and delirium. The temperature is likely to be elevated. The urine may contain albumin. The patient is restless and evinces a tendency to talkativeness; he is always busily engaged with his delusions. Sometimes the delirium is more violent. The victim is terror-stricken by hideous illusions and hallucinations. The figures of the wall-paper, and articles of furniture are converted into crawling reptiles; attendants, into demons. The patient is unable to sleep. Hypnotics may prove futile. There is little desire for food. That which is taken may be rejected. If recovery is to take place, sleep is gradually restored; else the patient is worn out and succumbs to exhaustion. Pneumonia, especially of the apices, is not an uncommon complication of alcoholism. The use of tobacco during convalescence is sufficient to renew the attack.

How is delirium tremens to be distinguished from acute mania?

The delirium of alcoholic intoxication may be maniacal, but it is associated with a history of alcoholism, while acute mania usually develops in a person with a psychopathic family history, and has probably been preceded by a prodromal period characterized by a change in manner, in disposition or in behavior. The character of the illusions and hallucinations differs in the two disorders. Delirium tremens usually subsides after sound sleep for a number of hours consecutively, and recovery is complete. Evidences of insanity persist after the attack of acute mania is at an end.

How are delirium tremens and cerebral meningitis to be differentiated?

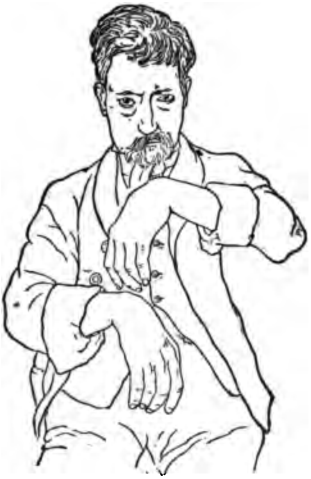
The distinction between delirium tremens and cerebral meningitis depends upon a knowledge of the cause, the history, the course, the symptoms and the termination of the two affections respectively. Delirium tremens is preceded by a history of alcoholism; meningitis, by a history of traumatism or infection. Delirium tremens lasts from a few days to a week; meningitis for a much longer time. In meningitis there are, and in delirium tremens there are not, muscular spasm, convulsions and sensory disturbance, followed by paralysis. The delirium and tremor of alcoholism are peculiar and characteristic. The temperature may be slightly elevated above the normal in delirium tremens, but meningitis is distinctly a febrile disease.

Plumbism.

What are the clinical manifestations of lead-poisoning?

Lead may gain entrance into the system and give rise to toxic manifestations under diverse circumstances. Thus saturnine intoxication occurs in those who work with the metal, as miners, color-grinders, painters, plumbers, type-founders, compositors. Lead may also be introduced into the system by means of drinking-water conveyed through lead-pipes, or with food prepared in leaden vessels, or by the use of hair-dyes, of cosmetics, or of snuff packed in lead-paper, or by the medicinal administration of lead in some form. The most common manifestation of plumbism is multiple neuritis.

FIG. 51.



Wrist-drop from lead poisoning. (Gowers.)

The symptoms vary in kind, degree, distribution and acuteness. The more common and the more characteristic are obstinate constipation, abdominal colic, wrist-drop, and a blue line on the gums. In addition, the nutrition is impaired and there is anemia. Among other symptoms are cramps in the legs, tremor, headache, convulsions, delirium and coma. The paralysis of the extensors of the wrist and fingers upon which the wrist-drop depends is bilateral, but does not involve the supinator longus. Sometimes, the intrinsic muscles of the hand atrophy; at times, too, the lower extremities are paralyzed. The knee-jerks may be enfeebled or lost; station may be unsteady; coördination may be deranged. Occasionally, symptoms of sensory derangement, laryngeal palsy, mental failure, melancholia, inequality of the pupils and impairment of vision are observed. The pupils may be small and fail to react to light. The optic nerve may undergo atrophy. Paralyzed muscles display the reaction of degeneration. Cardio-vascular-renal changes are of strikingly common occurrence. Arthritic manifestations and deposits indistinguishable from those of gout are not rare. If potassium iodide be administered, the urine may respond to chemic tests for lead.

How may lead-poisoning be confounded with cerebral tumor, and how is the differentiation to be made?

When the system has been impregnated with lead, there may be optic neuritis, headache, convulsions, delirium and palsy; but there are also a blue line on the gums; a history of exposure to the toxic metal; of intestinal colic; of obstinate constipation; and the palsy gives rise to wrist-drop.

Torticollis.

What is torticollis?

Torticollis or *wry-neck* is a condition in which, from shortening or from spasm of one or more muscles of the neck, the head is maintained in an abnormal position.

What are the varieties of torticollis?

There are two types of wry-neck. In one, as a result of traumatism, or of defective development, the muscle, usually the sterno-mastoid upon one side, is atrophied and shortened. The head is rotated to the side opposite to that of the affected muscle, which is conspicuous for its prominence. In the second variety of wry-neck, the muscles involved, most commonly the sterno-mastoid, the trapezius and the splenius, are in a state of active contraction, tonic or clonic, or alternately both. As a result, the head may be rotated or inclined to one side or retracted in over-extension, in accordance with the muscle or combination of muscles that participates in the spasm. Not infrequently, with the spasm of the muscles of the neck is also associated spasm of muscles of the arm or of the face.

Occupation-Neuroses.

What are the occupation-neuroses?

As the result of persistent and long-continued movement of a part in a constrained position, involving the activity of certain groups of muscles, spasmodic interference with the performance of the same movement may develop.

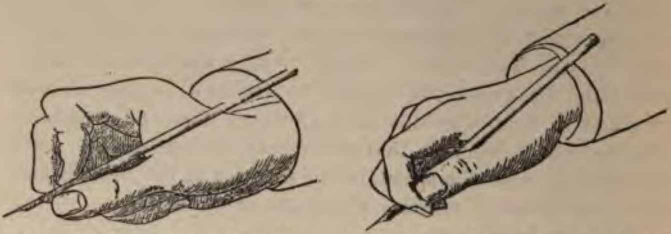
Thus, as a type of the disease, there is produced writers' cramp or scriveners' palsy. An analogous condition may be produced in telegraphers, shoemakers, piano-players, violinists, zither-players, seamstresses, smiths, painters, turners, watchmakers, knitters, engravers, masons, compositors, cigarette-makers, milkers, money-counters.

Writers' Cramp.

What are the symptoms of writers' cramp?

In those who write much, especially in such a way that the burden of the work falls upon the small muscles concerned, there sometimes develops a condition that renders writing impossible. Under such circumstances, as soon as an attempt to write is made, the fingers contract in spasm that may be painful, so that the act, if possible, is imperfectly accomplished. At other times, there may be tremor, and rarely weakness. In some cases, neuralgic pain is the most conspicuous symptom. In aggravated cases, the manifestations are not confined to the

Fig. 52.



Mode of holding the pen favorable to the development of writers' cramp. (Gowers.)

Mode of holding the pen when writing becomes difficult. (Gowers.)

act of writing, but may appear upon attempts to perform other movements. The onset of the symptoms is usually gradual, but occasionally it is sudden.

Chorea.

What are the etiologic elements concerned in the determination of an attack of chorea?

Chorea is most common at or about the age of puberty, and more so in girls than in boys. At Philadelphia, attacks are said to be more common in the spring of the year than at any other time. It has been observed that chorea is more common in white than in colored children. In many cases, an hereditary influence can be traced—directly, as chorea, or, more commonly, in the nature of other diseases of the nervous system, as epilepsy or insanity, or of rheumatism. Fright is frequently an exciting cause of the disease. Occasionally traumatism has preceded an attack. An obscure but well-determined causal relation exists between acute rheumatism, heart-disease and pregnancy, on the one hand, and chorea on the other.

What are the symptoms of chorea?

Chorea is a spasmodic neurosis, manifested by irregular, involuntary, incoördinated muscular movements, aggravated by excitement or observation. The movements cease during sleep, but they may be sufficiently violent to prevent sleep.

They are often more decided on one side of the body than on the other. Speech is often interfered with. A slight degree of muscular weakness exists. The electric irritability is usually increased; sometimes there are slight qualitative changes. Occasionally pain is felt in the parts affected. The temperature is, as a rule, slightly elevated. There is mental irritability or dulness of intellect. Anemia is often present.

At times, hemic heart-murmurs are heard; at other times, murmurs dependent upon organic disease, either antecedent or coincident, are heard; endocarditis is not an uncommon complication of chorea.

Chorea is a self-limited affection, the average duration of which is about ten weeks. It is said sometimes to involve the diaphragm, and to give rise to obstinate hiccough. It is thought that the irregularity of the action of the heart, sometimes observed in choreic children, is dependent upon involvement of the cardiac muscle; chorea of the larynx has also been seen.

So-called choreoid movement, athetosis, and mobile spasm, are often observed in cases of paralysis of cerebral origin.

What is hereditary chorea or Huntingdon's chorea?

Chorea sometimes appears in families, developing at the middle period of life, and possibly continuing until death, in hastening which it may play some part. The symptoms differ in no essential particular from those of the ordinary form of chorea.

Epilepsy.

What is the etiology of epilepsy?

Epilepsy is immediately dependent upon an irritable condition of the nervous system that must be referred to changes in the nerve-cells, beyond our present means of recognition. The disease is more common in females than in males. In a considerable proportion, a neurotic heredity can be traced. In many cases, no etiologic factor can be determined. The first attack usually occurs in early life, frequently in conjunction with dentition, or in association with rachitis. In other cases, it may be excited by mental influences, especially anxiety, by fails or blows, by acute disease, by the presence of parasites in the intestine, by menstrual derangement, by nasal polypi, by phimosis, by cicatrices, and by other sources of peripheral irritation.

When no source of irritation, direct or reflex, can be found, the disease is designated *essential* or *idiopathic*.

What are the symptoms of epilepsy?

Epilepsy is a paroxysmal neurosis, characterized by attacks of varying frequency, duration and severity, which may appear as transient loss of consciousness (*petit mal*, *minor epilepsy*) or may be preceded by a premonitory sensation or aura, followed successively by a cry, loss of consciousness, frothing at the mouth, tonic followed by clonic convulsions, headache, biting of the tongue and somnolence (*grand mal*, *major epilepsy*).

The attacks of petit mal may be so inconspicuous as to escape observation. There may be simply a vertiginous or other abnormal sensation, or an abrupt interruption in speech; or the grasp of an object held in the hand may be relaxed; or there may be a twitching of the muscles of the face.

When the seizure is preceded by an aura, this may be motor, sensory, visceral or psychic. In the attack the face becomes at first pallid, then flushed, and finally cyanotic; the pupil is dilated and insensible to light; urine and feces may be expelled involuntarily. The urine may contain a trace of albumin.

Mental deterioration ensues in a degree corresponding to the frequency of the attacks. Paroxysms may be succeeded by transient hemiplegia and aphasia, by the automatic performance of some act or by mania.

Sometimes a series of convulsions occurs, one attack scarcely ceasing before another begins, constituting the *status epilepticus*.

Nothing is known of the pathologic anatomy of epilepsy.

How are the epileptiform convulsions of cerebral disease to be distinguished from attacks of true epilepsy?

Epileptiform convulsions usually result from lesions involving the cortex cerebri. The most common of these lesions are softening from vascular occlusion, hemorrhage and new-growths. The convulsions occasioned are likely to be local in character and not at the onset attended with loss of consciousness; while the convulsions of epilepsy are usually general and simultaneous with loss of consciousness. In the intervals between epileptiform convulsions, some evidences of cerebral disease—hemiplegia, athetoid movements, choked discs—are likely to be present.

What are the distinctions between chorea and epilepsy?

Epilepsy occurs in paroxysms with intermissions, and may persist during the greater part of life; chorea is but a temporary, self-limited condition, the symptoms of which are constant during the continuance of the disease. The movements of chorea do not possess the violence of the convulsions of epilepsy; nor in chorea is consciousness lost or the tongue bitten.

How are the attacks of syncope and those of petit mal to be differentiated?

Syncope occurs under well-known circumstances that occasion temporary cerebral anemia; the pulse at the wrist is almost obliterated; loss of consciousness is of brief duration; the return to the normal condition takes place slowly. In case of *petit mal*, there have been previous attacks of a similar character or perhaps of major epilepsy; the loss of consciousness is sudden and transitory; the normal condition is soon resumed; there may have been slight muscular twitchings; or urine may have been passed involuntarily; or some automatic action is performed.

How is eclampsia to be distinguished from epilepsy?

By eclampsia is meant convulsions occurring incidentally and temporarily, as during dentition, or in connection with pregnancy and parturition. The relation of eclampsia to epilepsy and epileptiform convulsions is uncertain. The recognition of eclampsia and its distinction from epilepsy and epileptiform convulsions depend upon its incidental and transient occurrence in connection with one of the conditions that give rise to it.

How are the convulsions of uremia and those of epilepsy to be differentiated?

The convulsions of uremia are not unlike those of epilepsy. Their significance, however, depends upon their association with edema, a peculiar mustiness of the breath, the presence of albumin and tube-casts in the urine, and the existence of degenerative changes in the retina. If obtainable, the previous history may determine the diagnosis.

Hysteria.

What are the symptoms of hysteria?

Hysteria is a disease of the nervous system, more common in females than in males, and in early adult life than at any other period. In many cases a neuropathic heredity can be traced. The manifestations of the disease may be induced by mental emotion, anxiety, visceral disease and other disease. As a rule, they are sudden in onset and as sudden in disappearance. They may be dissipated by a profound mental impression. The symptoms are protean; some are continuous, others paroxysmal. Among the former are derangements of sensibility and motility, visceral, vaso-motor and cerebral disturbances; the paroxysmal symptoms include convulsive seizures. That which fundamentally characterizes the disease is a loss of self-control, a deficiency of will-power, a heightened self-consciousness, a tendency to exaggeration and a morbid desire for sympathy. The patient is moved to laughter or tears with undue readiness and without adequate cause. Duplicity and deception are practised, and there is an irresistible tendency to imitation. Sensation may be lost or heightened, or otherwise abnormal, locally, or upon one-half of the body, or in irregular distribution, with unsteady station and gait when the eyes are closed. Spinal tenderness is common. There is frequently a recurrent sensation of obstruction in the throat, as of a ball rising from the stomach, or of a sense of pharyngeal constriction—the so-called *globus hystericus.* At times, there is a feeling of intense pain in the head, such as might be occasioned by the driving in of a nail—the *clavus hystericus.* The special senses may be variously impaired. Motor palsy of diverse distribution may exist, occasioning paraplegia, hemiplegia or monoplegia, unattended with muscular wasting or alteration in the electric reactions or impairment of activity of the sphincters.

The reflexes are usually unaltered; in a number of cases, they are exaggerated. There may be aphonia, loss of the power of articulation, retention of urine, constipation, stammering. Contractures may take place and give rise to troublesome deformity. Laryngeal spasm may occasion distressing dyspnea, or a peculiar barking cough. Independently of any spasm, there may be increased frequency of respiration, with shortness of breath. Spasm of the pharynx or of the esophagus may entail the rejection of all food ingested. There may in addition be such repugnance to food that little is taken. On the other hand, the appetite may be strangely, even disgustingly perverted. There may be local flushing and perspiration. In females, menstruation may be deranged; ovarian tenderness is common. The paroxysmal attacks of convulsions are marked by tonic and clonic spasm, without loss of consciousness or biting of the tongue. The arms and legs are wildly thrown about by coördinated muscular activity—purposive movement. The patient may shriek and bite the lips. Opisthotonos and trismus may be developed. At times, there are evidences of profound psychic disturbance, as manifested by hallucinations and delusions, perhaps of an erotic nature. Lethargy and catalepsy are sometimes observed. The temperature may display decided irregularity.

How are hysterical paralysis and the cerebral paralysis of organic origin to be differentiated?

The palsy of hysteria, hemiplegic, paraplegic, diplegic or monoplegic, is usually atypical.

The face is rarely involved. Sensory derangement is common. The evidences of secondary descending degeneration: contractures and exaggeration of reflexes, are wanting. There may be constipation and retention of urine, but not incontinence.

While the onset may have been sudden, it will not have been apoplectic. Other symptoms of hysteria may make the character of the palsy clear, but the mere existence of the symptoms of hysteria should not obscure the possible simultaneous existence of organic disease.

How are the convulsions of hysteria and those of epilepsy to be differentiated?

An epileptic paroxysm takes place without special excitation, and sets in abruptly, often with a cry; an hysterical attack may be brought on by emotion, sets in gradually, and is attended with screaming. The convulsions of epilepsy pursue a regular sequence, and are associated with cyanosis, biting of the tongue, and insensibility of the iris to light; in the hysterical attack, the members are wildly thrown about, the pupil responds to stimulation, while the patient may bite the lips or hands or other persons or things. Involuntary extrusion of urine and feces may take place in epilepsy; but does not occur in hysteria.

Loss of consciousness is complete in epilepsy; consciousness is retained or perverted in hysteria, the paroxysm of which is characterized by talkativeness.

The epileptic attack lasts but a few minutes; the hysterical, ten minutes or longer. Opisthotonos is common in hysteria, exceptional in epilepsy.

An epileptic seizure may occur at any time and under any circumstances; hysterical attacks take place only in the presence of a second person. The hysterical patient is careful in the paroxysm to suffer no injury; the epileptic falls, whatever the attendant dangers, powerless to avert them.

An hysterical patient may, however, also be epileptic.

What are the distinctions between spastic paraplegia and hysterical paraplegia?

Spastic paraplegia develops at an age when hysteria is common; but in hysteria the reflexes are not exaggerated and the peculiar muscular spasm of lateral sclerosis does not occur, while sensory and emotional symptoms cannot escape observation.

How are acute myelitis and hysterical paraplegia to be differentiated?

Myelitis should not be overlooked because it occurs under circumstances amid which hysteria is to be expected. Exaggerated reflexes, trophic changes, wasting, derangement of sphincters and girdle-pain indicate the existence of more than hysteria.

How are the symptoms of hysteria and those of meningitis to be differentiated?

If symptoms of meningitis appear in an hysterical individual, their true significance may be overlooked. Convergent strabismus may be hysterical; but divergent strabismus always depends upon organic disease. There may be retention of urine in hysteria, but there is never incontinence. Trophic changes and continued elevation of temperature are evidences of the existence of something more than functional disease.

How are hysteria and cerebral tumor to be differentiated?

Hysteria may present any of the imitable symptoms of cerebral tumor, but in addition there will always be indubitable evidence of their nature. Optic neuritis or ptosis, of course, is not to be duplicated. Hysterical manifestations in association with cerebral symptoms should not be permitted to obscure the existence of organic disease.

Tetanus.

What are the symptoms of tetanus?

Tetanus is a spasmodic disorder dependent upon a specific infection contained in soil and introduced into the system through wounds or abrasions. It is manifested by painful rigidity of the head and jaw, soon progressing to trismus, and by stiffness of the tongue. In turn, the rigidity involves the muscles of the face (resulting in the *risus sardonicus*), the muscles of the trunk, the respiratory muscles and the diaphragm. To the tonic spasm of the muscles are added frequently recurring clonic exacerbations, which may be induced by external irritation.

The body may be arched in strong extension and supported only on the head and heels, constituting *opisthotonos*; it may be strongly arched forward (*emprosthotonos*), or laterally (*pleurosthotonos*), or it may be rigidly straight (*orthotonos*). The symptoms of tetanus set in at a variable period after inoculation—from a few hours to several days. The duration of the disease likewise varies from a few days to several weeks. Recovery is exceptional. Towards the close of life or even after death, the temperature may rise to an extraordinary height.

How are hysterical trismus and opisthotonos to be distinguished from tetanus?

Hysteria is an unruly disease, the symptoms of which are disorderly in appearance, while the symptoms of tetanus appear in fairly regular succession. Should trismus or opisthotonos develop as a manifestation of hysteria, it is likely to be associated with other symptoms of hysteria. The paroxysms remit and recur and do not go on to a fatal termination.

How is strychnine-poisoning to be distinguished from tetanus?

Strychnine-poisoning gives rise to some of the manifestations of tetanus. If trismus develops in strychnine-poisoning, it does so late; in tetanus, it is one of the first symptoms.

The convulsions of strychnine-poisoning are intermittent, but may be induced by external irritation; those of tetanus are continuous, with paroxysmal exacerbations; inquiry into the history of the case may elicit important evidence.

How are hemorrhage into the spinal membranes and tetanus to be differentiated?

Meningeal hemorrhage is sudden, tetanus gradual, in onset. Pain is a more prominent symptom in hemorrhage than in tetanus. Trismus is wanting in hemorrhage; it is characteristic of tetanus. Spasm is constant in tetanus, with exacerbations; intermittent in meningeal hemorrhage.

How are acute spinal meningitis and tetanus to be differentiated?

Meningitis sets in abruptly with a chill, followed by elevation of temperature; tetanus is of gradual development, usually after an injury, and is at the outset unattended with elevation of temperature. Trismus is characteristic of tetanus, but exceptional in meningitis.

The convulsions of tetanus are excited by slight peripheral irritation; the muscular contractions of meningitis are induced by efforts at movement.

Tetanus is far more commonly fatal than is meningitis. Motor and sensory impairment are common sequelæ of meningitis.

Tetany.

What is the etiology of tetany?

Tetany is most common in infancy and early adult life, when males are more prone to the disease than females. Occurring later in life, females are affected in larger proportion. In some cases, an hereditary influence can be made out. In many, diarrhea is an exciting cause. In others, the disease has been preceded by one of the acute infectious diseases. Pregnant or nursing women seem especially predisposed. The disease has been observed to develop in a considerable number of cases following removal of the thyroid gland. It has also been seen in association with dilatation of the stomach. In children it is often associated with rachitis, laryngismus stridulus, carpo-pedal spasm and convulsions. Other possible causes are exposure to cold and blows and injuries. Epidemics of the disease have been observed.

What are the symptoms of tetany?

Tetany is an affection characterized by muscular spasm, of symmetrical distribution, which usually begins and is most marked in the extremities.

The spasm may be continuous, remittent or intermittent.

A paroxysm may be induced by compression of the vessels and nerves of a part.

At the onset of the attack, there may be headache, vomiting, spinal pain, numbness and tingling. There may be moderate elevation of temperature.

In the intervals between paroxysms, the mechanical excitability and electric irritability of nerve and muscle are heightened. The disease may continue for a period ranging from a few days to several months.

Cases in which the spasm is intermittent are longer in duration than those in which the spasm is continuous.

How are tetany and tetanus to be distinguished?

The spasm of tetany is more likely than that of tetanus to be intermittent. Trismus is an early symptom of tetanus; if it occurs at all in tetany, it appears late. The participation of the extremities in the spasm is a feature of tetany.

Hydrophobia.

What are the clinical features of hydrophobia?

Hydrophobia results from the inoculation of man with rabies of animals. The infection is usually transmitted by the saliva of a rabid beast, as a dog, a cat, a fox, or a wolf, through a bite or a preëxisting wound. The period of incubation of hydrophobia is extremely variable, but is on an average from six to ten weeks. During this time, the primary wound may have healed, and no symptom have been present. Local pain may be perceived preceding the development of the disease proper, with the onset of which there are a sense of malaise, mental depression and slight difficulty in swallowing. Sleep is impaired and there may be respiratory spasm. The stomach now becomes intolerant, rejecting everything introduced. The muscular spasm extends and cutaneous hyperesthesia becomes manifest. External stimuli readily induce convulsions. The mental distress becomes intense. The temperature is elevated. Priapism may occur. Albumin and sugar are sometimes found in the urine. Ultimately, paralytic phenomena may supervene. Death may result from exhaustion, by reason of the inability to retain food, and the violence of the convulsions; from suffocation, as a result of respiratory spasm; or from heart-failure.

How are lyssophobia or pseudo-hydrophobia and true hydrophobia to be differentiated?

Persons who have been bitten by animals, rabid or not, develop a state of fear and dread, sometimes difficult to distinguish from true hydrophobia. Usually, however, the laryngeal and pharyngeal spasm characteristic of hydrophobia is wanting, while judicious moral assurance may cause a dissipation of the symptoms.

How are hydrophobia and tetanus to be differentiated?

In hydrophobia there is the history of a bite, with a long period of incubation; in tetanus of wound-infection with earth, and a short period of incubation. In hydrophobia, respiratory and pharyngeal spasm is an early manifestation; in tetanus, trismus is among the first symptoms. In the former all food is rejected; in the latter, if food can be introduced into the mouth, there is no difficulty of retention. Tetanus does not present the intensity of mental distress and disturbance encountered in hydrophobia.

Aural Vertigo—Labyrinthine Vertigo.

What is aural vertigo?

Menière's disease, *aural* or *labyrinthine vertigo*, is an affection dependent upon a pathologic condition of the terminal fibers of the auditory nerve in the labyrinth. The disease of the labyrinth may be inflammatory, gouty, syphilitic or degenerative. The symptoms occasioned are impairment of hearing, tinnitus aurium and vertigo. The last is aggravated in paroxysms, in which in addition there are nausea and vomiting, with pallor of the face and cold sweats. The patient may fall to the ground and the vertigo be so intense that he is temporarily unable to arise.

How are epilepsy and aural vertigo to be differentiated?

Epilepsy is, and aural vertigo is not, attended with muscular spasms and loss of consciousness. In the intervals between the paroxysms of aural vertigo some degree of dizziness persists and there are also impairment of hearing and tinnitus aurium, which are not accompaniments of epilepsy.

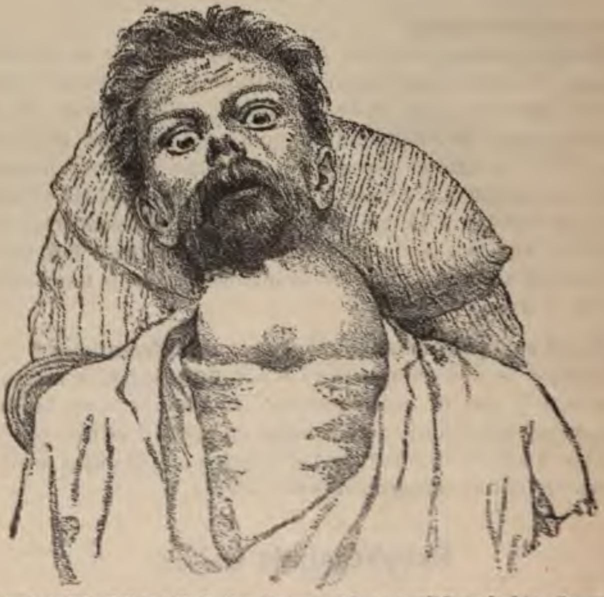
Exophthalmic Goiter.

What are the symptoms of exophthalmic goiter?

Exophthalmic goiter, also called *Graves's disease* and *Basedow's disease*, is an affection characterized by increased frequency of action and palpitation of the heart, protrusion of the eyeballs and enlargement of the thyroid gland. Its pathology is not yet established. It is clinically a vaso-motor ataxia, and has long been considered a disease of the sympathetic nervous system, especially of the cervical ganglia. There is growing evidence, however, that the disease is dependent upon a lesion of the floor of the fourth ventricle, with which many of the symptoms would harmonize.

The disease is more common in females than in males, and in adult life than at any other period. In some cases a neuropathic heredity can be traced. The exciting cause is usually mental emotion, shock, grief, anxiety.

Fig. 53.



Exophthalmic Goiter. Defective descent of the upper lids on looking down. (After Wilks.)

Palpitation of the heart, associated with or independent of an organic lesion, is one of the earliest symptoms. The action of the heart is increased both in frequency and in energy, and is often extremely irregular. The tachycardia and the palpitation may reach a high degree of intensity, the pulse sometimes exceeding 140 in the minute. There may also be evident pulsation in the arteries and a bruit in the vessels of the neck.

Anemia is often an early symptom. In the progress of the case, the eyes protrude and the thyroid gland becomes enlarged.

The exophthalmos and the goiter may each be symmetrical or unilateral; the protrusion and the enlargement, respectively, are not uncommonly greater upon the right side. In some cases, the upper lids are retracted, displaying the sclerotic, and do not follow the eyes when the glance is depressed. This lagging of the lids is called "Von Graefe's sign."

Over the enlarged thyroid gland a blowing systolic bruit is usually to be heard. The goiter frequently pulsates; it is at first usually elastic; ultimately it may become firm. The appearance of the goiter may be intermittent; when permanent, the enlargement may undergo spontaneous variation.

Among other symptoms are nystagmus, tremor of the extremities, an abnormal sense of heat, flushing and perspiration. It has been stated that the bodily electric resistance is diminished. Various cutaneous affections have been noted. In women, menstruation is commonly deranged.

In some cases, albuminuria, in others, diabetes has been observed. Hemoptysis, hematuria, hematemesis and other hemorrhages, and sometimes purpura, occur. Mental changes are not uncommon. Mania is an occasional complication.

Exophthalmic goiter is not directly fatal. Spontaneous recovery is possible. Temporary exacerbations take place.

What are the features distinguishing exophthalmic goiter from simple goiter?

Occasionally, the enlargement of the thyroid gland may be the first symptom of exophthalmic goiter observed. The bruit heard over the gland in exophthalmic goiter is wanting in the simple enlargement. Then, investigation will disclose the existence of palpitation and examination will reveal increased rapidity of the action of the heart, with the development, in the progress of the case, of protrusion of the eyes and the other symptoms of exophthalmic goiter. Even before the heart is much disturbed, there may be a tendency to flushing of the face, and occasionally red blood-cells may be found in the urine.

Cretinism.

What is cretinism?

Cretinism is a morbid condition, occurring endemically or sporadically and manifested by characteristic mental and physical abnormalities. The body is undersized; the head is broad, but shallow; the eyes are far apart and the nose is flat; the lips are thick; the tongue is sometimes enlarged; the hands are broad and spade-like; the skin is dry, rough and wrinkled; the aspect is that of age; the hair is straight and stiff; the intelligence is feeble, even to the degree of idiocy; masses of fat sometimes appear in the posterior triangles of the neck; the thyroid gland is often enlarged; it is sometimes wanting. In a number of cases premature ossification of the occipito-sphenoidal suture has been found. Some members of families in which cretinism existed have presented goiters.

Myxedema.

What are the clinical features of myxedema?

Myxedema is a peculiar condition, apparently more common in women than in men, in which a mucoid substance is found in the subcutaneous tissue in various parts of the body, in association with anemia, atrophy of the thyroid gland and mental impairment. The condition has been termed *sporadic cretinoidism* in distinction from sporadic cretinism.

The patient becomes dull, stupid and irritable; *speech* thick and hesitant; the *memory* enfeebled. *Movement* is awkward and clumsy, and there is undue readiness of fatigue. *Sensibility* is impaired, while abnormal sensations of heat and of chilliness are perceived. There may be visible flushing.

The *heart* is slow and feeble, the *respiration* sluggish, with breathlessness on exertion; the *temperature* is, as a rule, subnormal; the *appetite* is impaired, and taste is deranged; the *digestion* is enfeebled; the *nutrition* is poor; the *urine* is altered in quantity, and late in the disease may be albuminous.

The *face* is pallid and puffy, and of roundish ("full-moon") outline; the *expression* is dull and stupid; the *thyroid gland* is diminutive or absent. The *skin* is thickened, dry, rough, sometimes scaly, somewhat translucent in appearance, of a doughy consistence, but with a certain degree of elasticity; there is no edema and no pitting on pressure; perspiration is diminished. The *hands*, if affected, become square or "spade-shaped;" the *fingers* clubbed; and the *nails* brittle and distorted. The *hair* is dry and harsh and brittle, and may fall out.

In the progress of the case, mental and physical failure increase, irritability becomes marked, hallucinations develop, stupor sets in and death may take place in coma, from exhaustion or from uremia.

A condition resembling myxedema, occurring in circumscribed areas in various situations—*cachexia strumipriva*, has been induced in man and in animals by removal of the thyroid gland.

How are myxedema and scleroderma to be differentiated?

In a given case the diagnosis between myxedema and scleroderma may be exceedingly difficult. In scleroderma, however, the skin is brawny and indurated; not elastic and doughy, as in myxedema. In scleroderma, too, the thyroid gland remains unaffected, while the mental phenomena of myxedema are wanting. The histologic changes in the two conditions differ: in scleroderma, there is hyperplasia of the subcutaneous connective tissue; in myxedema, there is a mucoid deposit or degeneration.

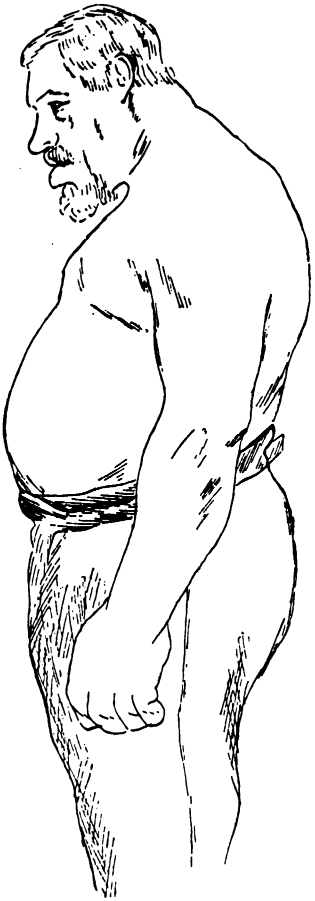
Akromegalia.

What is akromegalia?

Akromegalia or *Marie's disease* is a morbid condition that has been described by Marie, which is especially characterized by hypertrophy of the bones of the hands and feet and face.

Some of the fibro-cartilages, as those of the ear and larynx, also become enlarged. The related soft parts undergo corresponding enlargement. There develop decided weakness and slight muscular atrophy. The interosseous spaces become exaggerated. Owing to the enlargement of the inferior maxillary and the frontal bones, the face assumes a peculiarly elongated, elliptical outline. The hypertrophy of the nasal bones gives the nose a thickened appearance. The enlargement of the malar bones increases the normal temporal fossæ. The enlargement of the frontal sinuses and the projection of the superciliary ridges give the forehead a retreating appearance. The chin is prominent and the lower teeth project beyond the plane of the upper teeth. The tongue is enlarged and thickened. The lips and eyelids may also be thickened. The enlargement of the hands involves their width more than their length. The nails may present longitudinal striation. There are increased thirst, usually polyuria, and sometimes glycosuria. The appearance may be cachectic, the conjunctivæ icteric. There is commonly spinal curvature, usually a cervico-dorsal kyphosis, with a compensatory lumbar lordosis. The abdomen may be protuber-

FIG. 54.



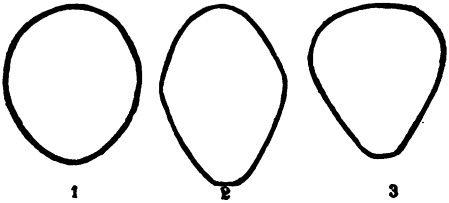
Akromegalia. (After Marie.)

ant. The stature is often increased. The skin may be pigmented. There may be severe spontaneous pain; headache is common. There may be somnolence. In some cases, varicose veins and hemorrhoids have been observed. In individual cases, there has been hemianopsia, limitation of the visual fields, blindness or deafness. In women, menstruation is usually deranged. The thyroid gland may be atrophied or hypertrophied. The disease appears most commonly in young adults and is chronic in course.

How does akromegalia differ from osteitis deformans, Paget's disease?

The disease called by Sir James Paget *osteitis deformans* more especially involves the long bones, which, while they become enlarged, also become distorted, with resulting deformities; akromegalia displays a predilection for the small bones of the hands and feet and face, and is unattended with distortion and deformity. In osteitis deformans, as a result of the *enlargement of the cranial bones*, the face presents a triangular outline, with the base above and the apex below; in akromegalia the *enlargement of the bones of the face* occasions a characteristic elongated, elliptical outline. In akromegalia there may be an increase in stature; in osteitis deformans there is more likely to be a diminution. The former appears early, between fifteen and thirty-five; the latter, later, after forty. Akromegalia may appear in several members of one family; osteitis deformans

FIG. 55.



Outline of the Face, 1, in myxedema; 2, in akromegalia; 3, in osteitis deformans. (Marie.)

does not. The invasion of akromegalia is symmetrical; that of osteitis deformans is indiscriminate.

How are akromegalia and myxedema to be differentiated?

The enlargement that appears in myxedema is dependent upon changes in the skin and subcutaneous soft structures; while the enlargement of akromegalia is due to a hyperplasia of bone. In the latter, the skin is healthy and mobile; in the former, it is thickened and adherent. In myxedema, in contrast to akromegalia, the hands and feet may be entirely uninvolved. In myxedema the face is almost round, "moon-shaped;" in akromegalia it is elliptical and elongated, with a prominent lower jaw, prominent malar processes and prominent nasal bones.

How is akromegalia to be discriminated from pulmonary hypertrophic osteo-arthropathy?

Under a name signifying "*hypertrophic disease of bones and joints in association with pulmonary lesion,*" Marie has discriminated a group of cases presenting, in connection with empyema, pleurisy, chronic bronchitis, tuberculous disease of the lungs, and possibly other disease of the lungs and their appendages, skeletal changes somewhat resembling those of akromegalia.

There are kyphosis, enlargement of the distal epiphyses of the bones of the forearm and of the leg, and, most characteristic, an enlargement in all directions of the distal phalanges of both fingers and toes, with the peculiar curvation of the nails that gives rise to the so-called Hippocratic finger. The lower jaw projects, but not so markedly as in akromegalia, and the other facial bones are not enlarged; on the contrary, the features are usually sharpened.

There is commonly dulness on percussion over the manubrium sterni, supposably dependent upon the persistence of the thymus gland. The thyroid gland may be wanting.

AKROMEGALIA.	PULMONARY OSTEO-ARTHROPATHY.
Stature heightened.	Stature not heightened.
Cervico-dorsal kyphosis.	Dorso-lumbar kyphosis.
Projection of abdomen.	No projection of abdomen.
Enlargement of face.	No enlargement of face.
Macroglossia.	Tongue not enlarged.

AKROMEGALIA.	PULMONARY OSTEO-ARTHROPATHY.
Hands broadened—spade-like.	Hands lengthened—not spade-like.
Digits uniformly enlarged.	Terminal phalanges enlarged.
Nail short, broad, flat, not reaching end of finger.	Hippocratic nail—curved, longitudinally striated, overlapping finger, " parrot-beak-like."
Epiphyseal enlargements uncommon.	Epiphyseal enlargements the rule.
Polyuria and disturbance of special senses.	Intra-thoracic disease.

INDEX.

ABORTIVE typhoid fever, 38
Abscess, cerebral, 322
Abscess, cerebral, differentiated from—
  cerebral meningitis, 323
  tumor of the brain, 323
Abscess, genito-urinary, 249
Abscess, lumbar, differentiated from—
  perityphlitis, 210
  typhlitis, 210
Abscess of the kidney, 268
Abscess of the kidney differentiated from perirenal abscess, 269
Abscess of the liver, 228
Abscess of the liver differentiated from—
  actinomycosis of the liver, 229
  carcinoma of the liver, 229
  hydatid cyst of the liver, 235
  occlusion of the biliary passages, 228
  pleuritic effusion, 156
Abscess of the mediastinum differentiated from aneurism of the aorta, 132
Abscess of the right ovary differentiated from—
  perityphlitis, 210
  typhlitis, 210
Abscess of the spleen differentiated from pleuritic effusion, 156
Abscess, perirenal, 269
Abscess, perirenal, differentiated from abscess of the kidney, 269
Abscess, pulmonary, differentiated from—
  bronchiectasis, 160
  pulmonary gangrene, 168
  pulmonary infarction, 167
  pulmonary tuberculosis, 176
Abscess, retro-pharyngeal, 194
Abscess, retro-pharyngeal, differentiated from membranous croup, 144
Acetone in the urine, tests for the presence of, 260
Acid, hydrochloric, free, in the gastric contents, tests for, 196
Acids, biliary, in the urine, 247
Actinomycosis, 83
Actinomycosis of the liver differentiated from abscess of the liver, 229
Action of the heart, 111
Acute alcoholism, 335
Acute anterior poliomyelitis, 290
Acute ascending paralysis, 291
Acute bronchitis, 157
Acute bulbar palsy, 296
Acute catarrhal pharyngitis, 188
Acute croupous pneumonia, 163
Acute dysentery, 206
Acute edema of the larynx, 139
Acute endocarditis, 128
Acute enteritis, 203, 204
Acute gastric catarrh, 197
Acute gastritis, 197
Acute general disease differentiated from cerebral meningitis, 316
Acute gout, 91
Acute hepatitis, 225
Acute indigestion, 197
Acute infective jaundice, 62
Acute intestinal catarrh, 203
Acute laryngitis, 138
Acute mania, 317, 336
Acute miliary tuberculosis, 177
Acute nasal catarrh, 136
Acute nephritis, 265
Acute pancreatitis, 241
Acute pericarditis, 127
Acute peritonitis, 219
Acute phlegmonous pharyngitis, 1

Acute pleurisy, 153
Acute rheumatism, 87
Acute synovitis, 88
Acute tuberculous laryngitis, 140
Acute tuberculous pharyngitis, 189
Acute yellow atrophy of the liver, 226
Addison's disease, 107
Ague-cake, 57
Ague, dumb, 56
Air-passages, obstruction of, differentiated from croup, 146
Akromegalia, 355
Akromegalia differentiated from—
  myxedema, 358
  osteitis deformans or Paget's disease, 357
  pulmonary hypertrophic osteoarthropathy, 358
Akromikria, 314
Albumin in urine, tests for, 250
Albuminuria, 250
Alcoholic intoxication differentiated from cerebral hemorrhage, 326
Alcoholism, acute, differentiated from sunstroke, 335
Ambulatory typhoid fever, 38
Amyloid disease of the liver, 231
Amyloid disease of the liver differentiated from—
  carcinoma of the liver, 232
  cirrhosis of the liver, 231
Amyloid kidney, 266
Amyotrophic lateral sclerosis, 292
Analgesic panaris, 312
Anamnesis, 19
Anemia, 86
Anemia, cerebral, 320
Anemia differentiated from cerebral tumor, 331
Anemia, idiopathic, 98
Anemia, infantile pseudo-leukemic, 103
Anemia, lymphatic, 101
Anemia of the spinal cord, 287
Anemia, pernicious, 98
Anemia, simple, 97
Anemia, splenic, 97
Anesthesia, 273
Anesthetic leprosy, 314
Aneurism, intracranial, 331
Aneurism of the aorta differentiated from abscess of the mediastinum, 132
Aneurism of the aorta differentiated from—
  aortic incompetency, 132
  dilatation of the auricle, 132
  intra-thoracic tumor, 131
Aneurism, thoracic, 131
Angina, 188
Angina pectoris, 118
Angina pectoris differentiated from—
  intercostal neuralgia, 119
  tachycardia, 117
Angina, ulcero-membranous, 192
Anthrax, 81
Aorta, aneurism of, 131, 132
Aortic incompetency or regurgitation, 124
Aortic obstruction, 123
Apex-beat of the heart, 109
Aphonia, hysterical, 139
Aphthous stomatitis, 184
Appendicitis, 209
Appendicitis, differential diagnosis of, 210
Arachnitis, 315
Ardent fever, 31
Arsenical poisoning differentiated from Asiatic cholera, 50
Arterio-capillary fibrosis, 132
Arthritic muscular atrophy, 299
Arthritis deformans, 94
Arthritis, rheumatoid, 94
Arthritis, syphilitic, 89
Ascaris lumbricoides, 217
Asiatic cholera, 49
Asphyxia differentiated from cerebral hemorrhage, 327
Asphyxia, local, 133
Asthma, 183
Asthma differentiated from—
  an asthmatoid condition, 183
  chronic edema of the larynx, 140
  laryngismus stridulus, 141
  paralysis of the diaphragm, 184
  whooping-cough, 184
Ataxia, 272, 273
Ataxia, Friedreich's, 304
Ataxia, locomotor, 300
Ataxic paraplegia, 303
Ataxic paraplegia, hereditary, 304
Atelectasis differentiated from catarrhal pneumonia, 163
Atrophy, muscular, arthritic, 299
Atrophy, muscular, progressive, 292, 293

Atrophy, muscular, progressive, differentiated from—
    acute anterior poliomyelitis, 295
    cervical pachymeningitis, 286
    chronic myelitis, 290
    simple idiopathic muscular atrophy, 299
    syringomelia, 312
Atrophy, muscular, simple idiopathic, 298
Atrophy, muscular, simple idiopathic, differentiated from—
    progressive muscular atrophy, 299
    pseudo-hypertrophic muscular paralysis, 299
Atrophy, yellow, acute, of the liver, 226
Atrophy, yellow, acute, of the liver differentiated from—
    catarrhal jaundice, 237
    phosphorus-poisoning, 227
    typhoid fever, 227
    yellow fever, 61
Aural vertigo, 351
Auricle, dilatation of, 120
Auscultation, 150
Auscultation of the heart-sounds, 110
Auscultatory percussion, 150
Autumnal catarrh, 137

BASEDOW'S disease, 351
Beri-beri, 276
Bile-duct, common, compression of, 237
Biliary acids in the urine, 247
Biliary calculi, 238
Biliary passages, carcinoma of, 239
Biliary passages, occlusion of, 228
Black tongue, 188
Bladder, neoplasms of, 262
Blood, 95
Blood-casts of the uriniferous tubules, 252
Blood in disease, 104
Boettger's test for the presence of sugar in the urine, 259
Bone, disease of, differentiated from neuritis, 276
Bothriocephalus latus, 216
Bowel, invagination or intussusception of, 212
Bowel, perforation of, in typhoid fever, 40
Break-bone fever, 76
Breathing, superior intercostal, differentiated from paralysis of the phrenic nerve, 281
Bronchial glands, tuberculosis of, 177
Bronchiectasis, 160
Bronchiectasis differentiated from—
    catarrhal pneumonia, 160
    pulmonary abscess, 160
    pulmonary gangrene, 160
    pulmonary tuberculosis, 176
Bronchitis, acute, 157
Bronchitis, acute, differentiated from acute miliary tuberculosis, 158
Bronchitis, capillary, 161
Bronchitis, capillary, differentiated from—
    acute miliary tuberculosis, 161
    catarrhal pneumonia, 162
Bronchitis, chronic, 158
Bronchitis, chronic, differentiated from—
    interstitial pneumonitis, 159
    pulmonary tuberculosis, 159
Bronchitis, plastic or fibrinous, 159
Bronchitis, putrid, 159
Bronchophony, 152
Broncho-pneumonia, 162
Brow-ague, 57

CACHEXIA, malarial, 56
Cachexia strumipriva, 355
Calculi, biliary, 238
Calculi, biliary, differentiated from—
    carcinoma of the biliary passages, 239
    catarrhal jaundice, 237
Calculus, renal, 264
Calculus, vesical, 262
Cancrum oris, 186
Capillary bronchitis, 161
Caput Medusæ, 230
Carcinoma of the biliary passages differentiated from biliary calculi, 239
Carcinoma of the cecum differentiated from—
    perityphlitis, 211
    typhlitis, 211
Carcinoma of the intestine, 213
Carcinoma of the liver, 232

Carcinoma of the liver differentiated from—
  abscess of the liver, 229
  amyloid disease of the liver, 232
  carcinoma of the omentum, 233
  carcinoma of the stomach, 233
Carcinoma of the omentum differentiated from—
  carcinoma of the liver, 233
  carcinoma of the stomach, 202
Carcinoma of the stomach, 200
Carcinoma of the stomach differentiated from—
  carcinoma of the liver, 233
  carcinoma of the omentum, 202
  carcinoma of the pancreas, 202
  chronic gastritis, 203
  sarcoma of the omentum, 202
  ulceration of the stomach, 201
Carcinoma, pulmonary, differentiated from pulmonary tuberculosis, 175
Cardiac impulse, 109
Caries of the vertebræ differentiated from tumor of thespinal cord, 311
Casts of the uriniferous tubules, 251
Catarrh, epidemic, 32
Catarrh, gastric, acute, 197
Catarrh, gastric, chronic, 198
Catarrh, gastro-intestinal, differentiated from pernicious malarial fever, 58
Catarrh, intestinal, acute, 203
Catarrh, intestinal, chronic, 206
Catarrhal croup, 142
Catarrhal fever, 32
Catarrhal jaundice, 237, 238
Catarrhal pharyngitis, acute, 188
Catarrhal pneumonia, 162
Catarrhal stomatitis, 184
Cavity, pulmonary, differentiated from pneumothorax, 182
Cecum, carcinoma of, 211
Cerebellar tumor, 302, 304
Cerebral abscess, 322
Cerebral anemia, 320
Cerebral congestion, 325
Cerebral disease differentiated from epilepsy, 342
Cerebral disease, paralysis of, differentiated from the paralysis of hysteria, 345
Cerebral embolism, 328
Cerebral functions, alterations in, in disease of the nervous system, 275
Cerebral hemorrhage, 323
Cerebral hyperemia, 321
Cerebral membranes, hemorrhage into, 318
Cerebral meningitis, 315
Cerebral softening, 327
Cerebral thrombosis, 329
Cerebral tumor, 330
Cerebritis, 322
Cerebritis differentiated from cerebral meningitis, 322
Cerebro-spinal fever, 45
Cerebro-spinal fever differentiated from—
  cerebral meningitis, 317
  tetanus, 47
  torticollis, 47
  typhoid fever, 48
  typhus fever, 47
  variola, 48
  yellow fever, 48
Cerebro-spinal sclerosis, 305
Cervical pachymeningitis, 286
Charbon, 81
Chickenpox, 73
Chickenpox differentiated from—
  measles, 73
  smallpox, 73
  varioloid, 73
Chlorosis, 98
Cholangitis, 236
Cholecystitis, 236
Cholera Asiatica, 49
Cholera Asiatica differentiated from cholera morbus, 50, 205
Cholera differentiated from pernicious malarial fever, 58
Cholera infantum, 206
Cholera infectiosa, 49
Cholera morbus, 205
Cholera morbus differentiated from cholera Asiatica, 50, 205
Cholera nostras, 205
Chorea, 340
Chorea differentiated from epilepsy, 343
Chorea, hereditary, 341
Chorea, Huntingdon's, 341
Chronic bronchitis, 158
Chronic dysentery, 208
Chronic edema of the larynx, 140
Chronic enteritis, 206
Chronic gastric catarrh, 198
Chronic gastritis, 198
Chronic gout, 92

Chronic interstitial nephritis, 266
Chronic intestinal catarrh, 206
Chronic laryngitis, 146
Chronic myelitis, 289
Chronic nephritis, 331
Chronic parenchymatous nephritis, 265
Chronic rheumatism, 90
Chronic tuberculosis of the larynx, 147
Chyluria, 253
Cirrhosis of the liver, 229
Cirrhosis of the liver differentiated from—
    amyloid disease of the liver, 231
    catarrhal jaundice, 238
    chronic perihepatitis, 236
Clavus hystericus, 344
Colic, hepatic, intestinal and renal, differential diagnosis of, 239
Colic, intestinal, differentiated from acute peritonitis, 220
Color of urine, 243
Coloring-matter, biliary, in urine, 246
Common membranous sore-throat, 192
Complications in disease, 22
Compression-myelitis differentiated from hemorrhage into the spinal cord, 310
Compression of the common bile-duct differentiated from catarrhal jaundice, 237
Compression, spinal, 309
Compression, spinal, differentiated from chronic myelitis, 310
Confluent smallpox, 70
Congenital spastic paraplegia, 319
Congestion, cerebral, differentiated from cerebral hemorrhage, 325
Congestion of the liver, 224
Congestion, pulmonary, differentiated from croupous pneumonia, 166
Constitution of urine, 242
Constitutional signs, 19
Consumption, galloping, 169, 174
Continued fever, 30
Convulsion, 273
Cord, spinal, anemia of, 287
Cord, spinal, hyperemia of, 287
Coryza, 136
Coryza differentiated from—
    hay-fever, 138
    influenza, 137
Cramp, writers', 339
Cretinism, 354
Cretinoidism, sporadic, 354
Croup, catarrhal, 142
Croup differentiated from obstruction of the air-passages, 145
Croup, membranous, 143
Croup, membranous, differentiated from—
    diphtheria, 80
    retro-pharyngeal abscess, 144
    spasmodic croup, 144
    whooping-cough, 145
Croup, spasmodic, 142
Croup, spasmodic, differentiated from—
    edema of the larynx, 143
    laryngismus stridulus, 142
    membranous croup, 144
Croupous enteritis, 205
Croupous pneumonia, 163
Cystitis, 249, 261
Cystitis differentiated from—
    nephritis, 261
    pyelitis, 263

DATA for diagnosis, 24
Deep reflexes, 274
Degeneration, fatty, of the heart, 121
Degeneration of the diaphragm, 281
Degeneration, reaction of, 274
Delirium tremens, 335
Delirium tremens differentiated from—
    acute mania, 336
    cerebral meningitis, 336
Dengue, 76
Dengue differentiated from—
    influenza, 77
    scarlet fever, 77
Dextrocardia, 115
Diabetes insipidus, 256
Diabetes insipidus differentiated from—
    chronic interstitial nephritis, 257
    diabetes mellitus, 261
    hysterical polyuria, 257
Diabetes mellitus, 257
Diabetes mellitus differentiated from—
    diabetes insipidus, 261
    Morvan's disease, 314
Diacetic acid in the urine, tests for the presence of, 260

Diaceturia, 260
Diagnosis, 17
Diagnosis by exclusion, 21
Diagnosis by the historical or empirical method, 20
Diagnosis by the inductive method, 20
Diagnosis by the method of pathological association, 20
Diagnosis, data for, 24
Diagnosis, direct, 20
Diagnosis, discriminative or differential, 21
Diagnosis, physical, 147
Diaphragm, degeneration of, differentiated from paralysis of the phrenic nerve, 281
Diaphragm, inflammation of, differentiated from paralysis of the phrenic nerve, 281
Diaphragm, paralysis of, 184
Diaphragmatic hernia, 182
Differential diagnosis, 21
Digestion, duration of, 195
Digestive system, 184
Dilatation of the auricle differentiated from aneurism of the aorta, 132
Dilatation of the heart, 120
Dilatation of the heart differentiated from—
  fatty degeneration of the heart, 121
  hypertrophy of the heart, 120
  pericardial effusion, 121
Dilatation of the stomach, 199
Diphtheria, 77
Diphtheria differentiated from—
  common membranous sore-throat, 192
  gangrenous sore-throat, 193
  membranous croup, 80
  scarlet fever, 78
  stomatitis, 80
  tonsillitis, 79
Diphtheroid throat, 192
Diplegia, 272
Direct diagnosis, 20
Discrete smallpox, 70
Discriminative diagnosis, 21
Disseminated neuritis, 276
Disseminated sclerosis, 305
Distention of the gall-bladder differentiated from hydatid cyst of the liver, 235
Drug-effects, 23
Dumb ague, 56
Duplication of the heart-sounds, 111
Duration of digestion, 195
Dysentery, acute, 206
Dysentery, acute, differentiated from—
  acute enteritis, 207
  intussusception of the bowel, 207
  typhoid fever, 207
Dysentery, chronic, 208

ECLAMPSIA differentiated from epilepsy, 343
Edema of the larynx, acute, 139
Edema of the larynx, chronic, 140
Edema of the larynx differentiated from—
  asthma, 140
  spasmodic croup, 143
Edema, pulmonary, differentiated from pneumonia, 166
Efficiency of the heart, 112
Effusion, pericardial, differentiated from—
  dilatation of the heart, 121
  pleuritic effusion, 157
Effusion, pleural, differentiated from—
  pneumothorax, 182
  pulmonary emphysema, 181
Effusion, pleuritic, differentiated from—
  abscess of the liver, 156
  abscess of the spleen, 156
  hydatid cyst of the liver, 155
  hydrothorax, 157
  pericardial effusion, 157
Egophony, 152
Electric reactions, alterations in, in disease, 274
Embolism, cerebral, 328
Embolism, cerebral, differentiated from cerebral hemorrhage, 328
Emphysema, pulmonary, 179
Emphysema, pulmonary, differentiated from—
  pleural effusion, 181
  pneumothorax, 180
Empirical method, diagnosis by, 20
Emprosthotonos, 347
Empyema, 153
Empyema, pulsatory, 153
Endemic hematuria, 256

Endocardial murmurs, 126
Endocarditis, 128
Endocarditis, acute, differentiated from—
  acute pericarditis, 129
  valvular disease of the heart, 128
Endocarditis, mycotic, 129
Enteric fever, 35
Enteritis, acute, differentiated from—
  acute dysentery, 207
  acute peritonitis, 220
Enteritis, catarrhal, acute, differentiated from typhoid fever, 204
Enteritis, chronic, 206
Enteritis, croupous, 205
Enteritis, membranous, 205
Enteritis, mucous, acute, 203
Eosinophile leukocytes, 96
Ephemeral fever, 31
Epidemic catarrh, 32
Epidemic cerebro-spinal meningitis, 45
Epilepsy, 341
Epilepsy differentiated from—
  aural vertigo, 351
  cerebral disease, 342
  cerebral tumor, 331
  chorea, 343
  eclampsia, 343
  hysteria, 345
  syncope, 343
  uremia, 267, 343
Epilepsy, essential, 342
Epilepsy, idiopathic, 342
Epilepsy, major, 342
Epilepsy, minor, 342
Epistaxis, 136
Epithelial casts of the uriniferous tubules, 252
Equinia, 80
Erysipelas, 74
Erysipelas differentiated from—
  erythema, 75
  herpes zoster, 75
  scarlet fever, 74
  smallpox, 75
Erythema differentiated from erysipelas, 75
Esophagismus, 195
Esophagus, 194
Esophagus, stricture of, 194
Essential epilepsy, 342
Essential fever, 30
Examination of urine, 242
Exclusion, diagnosis by, 21
Exophthalmic goiter, 351
Exophthalmic goiter differentiated from—
  simple goiter, 353
  tachycardia, 117

FACIAL nerve, paralysis of, 279
Family history, 19
Farcy, 80
Fatty casts of the uriniferous tubules, 252
Fatty degeneration of the heart differentiated from dilatation of the heart, 121
Fatty infiltration of the liver, 230
Fatty liver, 230
Fatty metamorphosis of the liver, 230
Febricula, 31
Febrile temperature, 28
Fehling's test for the presence of sugar in the urine, 259
Fermentation-test for the presence of sugar in the urine, 260
Fever, 27
Fever, ardent, 31
Fever, break-bone, 76
Fever, catarrhal, 32
Fever, cerebro-spinal, 45
Fever, continued, 30
Fever, enteric, 35
Fever, ephemeral, 31
Fever, essential, 30
Fever, hemorrhagic malarial, 56
Fever, hepatic intermittent, 239
Fever, intermittent, 30
Fever, malarial intermittent, 54
Fever, malarial remittent, 55
Fever of suppuration, 57
Fever, pernicious malarial, 56
Fever, rag-weed, 137
Fever, relapsing, 51
Fever, remittent, 30
Fever, rheumatic, 87
Fever, scarlet, 65
Fever, simple continued, 31
Fever, specific, 30
Fever, splenic, 81
Fever, symptomatic, 30
Fever, syphilitic, 59
Fever, thermic, 32, 334
Fever, typhoid, 35

Fever, typhus, 43
Fever, yellow, 59
Fibrinous bronchitis, 159
Fibroid phthisis, 160
Fibrosis, arterio-capillary, 132
Filariosis, 253
Floating kidney, 264
Floating liver, 223
Floating spleen, 241
Florid phthisis, 169, 174
Follicular tonsillitis, 191
Foot-and-mouth disease, 84
Fremitus, vocal or tactile, 148
French measles, 68
Frequency of action of the heart, 110
Friedreich's ataxia, 304
Frost-bite differentiated from Raynaud's disease, 134
Functional heart-murmurs, 114
Functional stricture of the esophagus, 195

GALL-BLADDER, distention of, 235
Galloping consumption, 169, 174
Gangrene, pulmonary, 168
Gangrene, pulmonary, differentiated from—
  bronchiectasis, 160
  pulmonary abscess, 168
  pulmonary tuberculosis, 169
Gangrene, symmetrical, 133
Gangrenous pharyngitis, 193
Gangrenous stomatitis, 186
Gastralgia, 196
Gastrectasia, 199
Gastric catarrh, acute, 197
Gastric catarrh, chronic, 198
Gastric contents, free hydrochloric acid in, tests for, 196
Gastric hemorrhage, 175
Gastric ulcer, 199
Gastritis, acute, 197
Gastritis, acute, differentiated from—
  acute peritonitis, 219
  intestinal obstruction, 198
  the gastric symptoms of acute febrile disease or of cerebral disease, 197
Gastritis, chronic, 198
Gastritis, chronic, differentiated from—
  carcinoma of the stomach, 203
  gastric ulcer, 200
Gastrodynia, 196
Gastro-enteritis, 204
Gastro-intestinal catarrh, 58
General paralysis of the insane, 332
General signs, 19
Genito-urinary apparatus, 242
German measles, 68
Glanders, 80
Glanders differentiated from—
  smallpox, 81
  syphilis, 81
Globus hystericus, 344
Glossanthrax, 187
Glossitis, 187
Glosso-labio-laryngeal paralysis, 292
Glycosuria, 257
Goiter, exophthalmic, 351
Goiter, simple, differentiated from exophthalmic goiter, 353
Gonorrheal synovitis, 88
Gout, acute, 91
Gout, acute, differentiated from acute rheumatism, 92
Gout, chronic, 92
Gout differentiated from rheumatoid arthritis, 95
Grand mal, 342
Granular casts of the uriniferous tubules, 252
Graves's disease, 351
Green-sickness, 98
Grippe, la, 32

HAIRY tongue, 188
Hay-asthma, 137
Hay-fever, 137
Hay-fever differentiated from coryza, 138
Headache differentiated from migraine, 284
Headache, sick, 284
Heart, 109
Heart, action of, 111
Heart, action of, intermission of, 111
Heart, apex-beat of, 109
Heart-clot, 130
Heart, dilatation of, 120
Heart, efficiency of, 112
Heart, fatty degeneration of, 121
Heart, functional disturbance of, 116
Heart, hypertrophy of, 119
Heart, irritable, 117
Heart, malformation of, 115
Heart-murmurs, 112

Heart-murmurs, organic, differentiated from functional, 114
Heart-sounds, auscultation of, 110
Heart, valvular disease of, 121
Heat-exhaustion, 334
Heat-exhaustion differentiated from heat-fever or sunstroke, 335
Heat-fever, 334
Heat-stroke, 334
Hematuria, 254
Hematuria, endemic, 256
Hematuria, malarial, differentiated from paroxysmal hemoglobinuria, 255
Hemicrania, 284
Hemiplegia, 272
Hemocytometry, 95
Hemoglobinometry, 96
Hemoglobinuria, 254
Hemoglobinuria, paroxysmal, 255
Hemoglobinuria, paroxysmal, differentiated from malarial hematuria, 255
Hemophilia, 106
Hemorrhage, cerebral, 323
Hemorrhage, cerebral, differentiated from—
  alcoholic intoxication, 326
  asphyxia, 327
  cerebral congestion, 325
  cerebral embolism, 328
  cerebral thrombosis, 329
  opium-poisoning, 326
  primary lateral sclerosis, 303
  sunstroke, 335
  syncope, 325
  uremia, 326
Hemorrhage, gastric, differentiated from pulmonary hemorrhage, 175
Hemorrhage into the cerebral membranes, 318
Hemorrhage into the spinal membranes, 286
Hemorrhage into the spinal membranes differentiated from—
  spinal hemorrhage, 308
  spinal meningitis, 287
  tetanus, 348
Hemorrhage, pulmonary, differentiated from gastric hemorrhage, 175
Hemorrhage, spinal, 308
Hemorrhage, spinal, differentiated from—
  acute myelitis, 289
  compression-myelitis, 310

Hemorrhage, spinal, differentiated from—
  meningeal hemorrhage, 308
  spinal meningitis, 308
Hemorrhagic malarial fever, 56
Hemorrhagic pachymeningitis, 315
Hemorrhagic smallpox, 71
Hepatic colic, 239
Hepatic intermittent fever differentiated from malarial intermittent fever, 239
Hepatitis, acute, 225
Hepatitis, acute, differentiated from—
  malarial cachexia, 225
  portal phlebitis, 225
Hepatitis, interstitial, 229
Hereditary ataxic paraplegia, 304
Hereditary chorea, 341
Hernia, diaphragmatic, differentiated from pneumothorax, 182
Herpes of the pharynx, 192
Herpes zoster differentiated from erysipelas, 75
Herpetic sore-throat, 192
Herpetic tonsillitis, 192
Hip-joint, disease of, differentiated from sciatica, 278
Hippuric acid in urine, 245
Historical method, diagnosis by, 20
History, family, 19
History, previous, 19
Hodgkin's disease, 101
Humanized virus, 71
Huntingdon's chorea, 341
Hyaline casts of the uriniferous tubules, 252
Hydatid cyst, 84
Hydatid cyst of the kidney, 271
Hydatid cyst of the kidney differentiated from hydronephrosis, 271
Hydatid cyst of the liver, 234
Hydatid cyst of the liver differentiated from—
  abscess of the liver, 235
  distention of the gall-bladder, 235
  pleuritic effusion, 155
Hydatid disease, 84
Hydremia, 96
Hydrocephalus, 318
Hydrochloric acid in the gastric contents, 196
Hydronephrosis, 270

Hydronephrosis differentiated from cyst of the kidney, 271
Hydropericardium, 130
Hydrophobia, 350
Hydrophobia differentiated from—
  pseudo-hydrophobia, 350
  tetanus, 351
Hydrothorax differentiated from pleuritic effusion, 157
Hyperemia, cerebral, 321
Hyperemia of the spinal cord, 287
Hyperesthesia, 273
Hyperpyrexia, 28
Hypertrophic pachymeningitis, 311, 312
Hypertrophy of the heart, 119
Hypertrophy of the heart differentiated from dilatation of the heart, 120
Hypesthesia, 273
Hysteria, 344
Hysteria differentiated from—
  acute meningitis, 347
  cerebral tumor, 347
  epilepsy, 345
  tetanus, 348
Hysterical aphonia differentiated from acute laryngitis, 139
Hysterical paralysis differentiated from the paralysis of cerebral disease, 345
Hysterical paraplegia differentiated from—
  acute myelitis, 346
  spastic paraplegia, 346
Hysterical polyuria, 257

IDIOPATHIC anemia, 98
Idiopathic epilepsy, 342
Idiosyncratic coryza, 137
Immediate percussion, 149
Impulse, cardiac, 109
Inadequacy, renal, 263
Incompetency, aortic, 124
Incompetency, aortic, differentiated from aneurism of the aorta, 132
Incompetency, mitral, 122
Incompetency, pulmonary, 125
Incompetency, tricuspid, 125
Indigestion, acute, 197
Inductive method, diagnosis by, 20
Infantile pseudo-leukemic anemia, 103
Infantile spastic paraplegia, 319
Infarction, pulmonary, differentiated from—
  catarrhal pneumonia, 163
  croupous pneumonia, 167
  pulmonary abscess, 167
Inflammation of the diaphragm, 281
Influenza, 32
Influenza differentiated from—
  coryza, 137
  dengue, 77
Inquiry, 24
Insane, general paralysis of, 332
Insolation, 32, 334
Inspection, 109, 134, 147
Insular sclerosis, 305
Intercostal neuralgia, 119, 155
Intermission of the heart's action, 111
Intermittent fever, 30
Intermittent fever, hepatic, 239
Intermittent fever, malarial, 54
Interstitial hepatitis, 229
Interstitial myocarditis, 130
Interstitial pnemonitis, 179
Interstitial nephritis, chronic, 266
Intestinal catarrh, acute, 203
Intestinal catarrh, chronic, 206
Intestinal colic, 220, 239
Intestinal obstruction, 211
Intestinal parasites, 214
Intestines, 203
Intestines, carcinoma of, 213
Intoxication, alcoholic, 326
Intracranial aneurism, 331
Intra-thoracic tumor, 131, 156
Intussusception of the bowel, 212
Intussusception of the bowel differentiated from—
  acute dysentery, 207
  obstruction of the bowel, 213
  typhlitis, 213
Invagination of the bowel, 212
Inverse temperature, 28
Irritable heart, 117
Irritable heart differentiated from tachycardia, 118

JAIL-FEVER, 43
Jaundice, catarrhal, differential diagnosis of, 236
Jaundice, catarrhal, differentiated from—
  acute yellow atrophy of the liver, 237

Jaundice, catarrhal, differentiated from—
  biliary calculus, 237
  cirrhosis of the liver, 238
  compression of the common bile-duct, 237
  malarial fever, 237
  malignant disease, 237
  pylephlebitis, 237
  yellow fever, 237
June-cold, 137

KAK-KÉ, 276
Kidney, abscess of, 268
Kidney, amyloid, lardaceous or waxy, 266
Kidney, floating, 264
Kidney, hydatid cyst of, 271
Kidney, malignant disease of, 270
Kidney, tuberculosis of, 269

LABYRINTHINE vertigo, 351
Lacunal tonsillitis, 191
Landry's paralysis, 291
Lardaceous kidney, 266
Larval paludism, 57
Laryngeal neoplasms differentiated from chronic laryngitis, 146
Laryngeal vertigo, 142
Laryngismus stridulus, 141
Laryngismus stridulus differentiated from—
  asthma, 141
  catarrhal croup, 142
  whooping-cough, 146
Laryngitis, acute, 138
Laryngitis, acute, differentiated from hysterical aphonia, 139
Laryngitis, chronic, 146
Laryngitis, chronic, differentiated from laryngeal neoplasms, 146
Laryngitis differentiated from—
  parotiditis, 139
  pharyngitis, 138
  tonsillitis, 139
Laryngitis, pseudo-membranous, 143
Laryngitis, tuberculous, acute, 140
Laryngoscopy, 135
Larynx, edema of, 139
Larynx, tuberculosis of, chronic, 147
Lateral sclerosis, primary, 302
Lead-poisoning, 337
Lead-poisoning differentiated from cerebral tumor, 338
Lead-poisoning, neuritis of, differentiated from neuritis of the musculo-spiral nerve, 282
Leprosy, anesthetic, differentiated from Morvan's disease, 314
Leptomeningitis, 135
Leucin, 247
Leukemia, 96, 99
Leukemia differentiated from pseudo-leukemia, 102
Leukocythemia, 96, 99
Leukocytosis, 96, 99
Leukoplakia lingualis s. buccalis, 187
Lipuria, 253
Lithemia, 93
Liver, 222
Liver, abscess of, 228
Liver, actinomycosis of, 229
Liver, amyloid disease of, 231
Liver, atrophy of, acute yellow, 226
Liver, carcinoma of, 232
Liver, cirrhosis of, 229
Liver, congestion of, 224
Liver, fatty, 230
Liver, floating, 223
Liver, hydatid cyst of, 234
Liver, malignant disease of, differentiated from chronic peritonitis, 222
Lobar pneumonia, 163
Lobular pneumonia, 162
Local asphyxia, 133
Local signs, 19
Locomotor ataxia, 300
Lumbar pachymeningitis, 301
Lumbricoid worms, 217
Lymphadenoma, 101
Lymphatic anemia, 101
Lymph-glands, tuberculosis of, 102
Lymphoma, malignant, 101
Lyssophobia, 350

MAJOR epilepsy, 342
Malaria, masked, 57
Malarial cachexia, 56
Malarial cachexia differentiated from chronic hepatitis, 225
Malarial diseases, 52
Malarial fever differentiated from—
  catarrhal jaundice, 237
  pulmonary tuberculosis, 177

Malarial fever differentiated from—
  syphilitic fever, 59
  the fever of suppuration or of septic infection, 57
  typhoid fever, 59
Malarial fever, hemorrhagic, 56
Malarial fever, hemorrhagic, differentiated from—
  paroxysmal hemoglobinuria, 58
  yellow fever, 61
Malarial fever, pernicious, 56
Malarial fever, varieties of, 53
Malarial hematuria, 255
Malarial intermittent fever, 54
Malarial intermittent fever differentiated from hepatic intermittent fever, 239
Malarial paroxysm, 54
Malarial remittent fever, 55
Malarial remittent fever differentiated from yellow fever, 60
Malformation of the heart, 115
Malignant disease differentiated from catarrhal jaundice, 237
Malignant disease of the kidney, 270
Malignant disease of the liver, 222
Malignant lymphoma, 101
Malignant pustule, 81
Malignant smallpox, 71
Mania, acute, differentiated from—
  cerebral meningitis, 317
  delirium tremens, 336
Marie's disease, 355
Masked malaria, 57
Measles, 63
Measles differentiated from—
  chickenpox, 73
  purpura, 106
  Rötheln, 69
  scarlet fever, 68
  smallpox, 72
  typhus fever, 65
Mediastinum, abscess of, 132
Mediate percussion, 149
Megalocytes, 96
Mellituria, 257
Membranes, spinal, hemorrhage into, 286
Membranous croup, 148
Membranous enteritis, 205
Membranous sore-throat, common, 192
Menière's disease, 351
Meningitis, acute, differentiated from hysteria, 347
Meningitis, acute, differentiated from sunstroke, 335
Meningitis, cerebral, 315
Meningitis, cerebral, differentiated from—
  acute general disease, 316
  acute mania, 317
  cerebral abscess, 323
  cerebritis, 322
  cerebro-spinal fever, 317
  delirium tremens, 336
Meningitis, cerebral, simple, differentiated from tuberculous meningitis, 316
Meningitis, spinal, 285
Meningitis, spinal, acute, differentiated from—
  acute myelitis, 288
  tetanus, 348
Meningitis, spinal, differentiated from—
  hemorrhage into the spinal meninges, 287
  rheumatism of the muscles of the back, 285
  spinal hemorrhage, 308
Meningitis, tuberculous, differentiated from simple cerebral meningitis, 316
Mensuration, 148
Mercurial stomatitis, 186
Microcytes, 96
Migraine, 284
Migraine differentiated from ordinary headache, 284
Miliary tuberculosis, acute, 177
Minor epilepsy, 342
Mitral incompetency or regurgitation, 122
Mitral obstruction, 123
Mollities ossium, 108
Mollities ossium differentiated from rachitis, 109
Monoplegia, 272, 273
Moore's test for the presence of sugar in the urine, 259
Morbilli, 63
Morvan's disease, 312
Morvan's disease differentiated from—
  anesthetic leprosy, 314
  diabetes, 314
  Raynaud's disease, 314
  scleroderma, 313
  syphilis, 314

Morvan's disease differentiated from syringomyelia, 314
Motion, 272
Mouth, 184
Mucous enteritis, 203
Muguet, 185
Multiple neuritis, 276
Multiple sclerosis, 305
Mumps, 188
Murmurs, endocardial, 126
Murmurs, organic, differentiated from functional heart-murmurs, 114
Musculo-spiral nerve, paralysis of, 281
Musculo-spiral neuritis differentiated from the neuritis of lead-poisoning, 282
Myalgia, 90
Mycotic endocarditis, 129
Myelitis, 288
Myelitis, acute, differentiated from—
  acute anterior poliomyelitis, 291
  acute ascending paralysis, 292
  acute spinal meningitis, 288
  chronic myelitis, 287
  hysterical paraplegia, 346
  multiple neuritis, 279
  spinal hemorrhage, 289
Myelitis, chronic, 289
Myelitis, chronic, differentiated from—
  acute myelitis, 289
  compression of the cord, 310
  lateral sclerosis, 290
  primary lateral sclerosis, 302
  progressive muscular atrophy, 290
  spinal pachymeningitis, 290
  syringomyelia, 312
  tumor of the spinal cord, 311
Myocarditis, 130
Myocarditis, interstitial, 130
Myotonia congenita, 300
Myxedema, 354
Myxedema differentiated from—
  akromegalia, 358
  scleroderma, 355

NASAL catarrh, 136
Nasal respiration, difficulty of, 135
Nasal septum, ulceration or erosion of, 136
Negative signs, 23
Neoplasms, laryngeal, 146
Neoplasms of the bladder, 262
Nephritis, acute, 265
Nephritis, chronic, differentiated from cerebral tumor, 331
Nephritis, chronic interstitial, 266
Nephritis, chronic interstitial, differentiated from diabetes insipidus, 257
Nephritis, chronic parenchymatous, 265
Nephritis differentiated from cystitis, 261
Neuralgia, 283
Neuralgia differentiated from—
  neuritis, 276, 283
  subacute rheumatism, 89
Neuralgia, intercostal, differentiated from—
  acute pleurisy, 155
  angina pectoris, 119
Neuralgia of the stomach, 196
Neuralgia, sciatic, differentiated from sciatica, 278
Neuralgia, trifacial or trigeminal, 283
Neuritis, 275
Neuritis, diagnosis of, 276
Neuritis differentiated from—
  disease of bone, 276
  neuralgia, 276, 283
  subacute rheumatism, 276
Neuritis, multiple or disseminated, 276
Neuritis, multiple, differentiated from—
  acute anterior poliomyelitis, 291
  acute ascending paralysis, 292
  acute myelitis, 277
  posterior spinal sclerosis, 301
  spinal pachymeningitis, 277
Neuritis, musculo-spiral, 282
Neuromata, 282
Neuromata, diagnosis of, 282
Nigrities, 188
Noma, 186
Nutrition, alterations in, in disease of the nervous system, 271

OBJECTIVE symptoms, 17
Observation, 24
Obstruction, aortic, 123
Obstruction, intestinal, 211

Obstruction, intestinal, acute, differentiated from acute peritonitis, 220
Obstruction, intestinal, differentiated from acute gastritis, 198
Obstruction, mitral, 123
Obstruction of the air-passages, 146
Obstruction of the bowel differentiated from intussusception, 213
Obstruction, pulmonary, 125
Obstruction, tricuspid, 125
Occlusion of the biliary passages differentiated from abscess of the liver, 228
Occupation-neuroses, 339
Odor of urine, 242, 243
Oligemia, 96
Oligochromemia, 96
Oligocythemia, 96
Omentum, carcinoma of, 202, 233
Omentum, sarcoma of, 202
Opisthotonos, 347
Opium-poisoning differentiated from cerebral hemorrhage, 326
Organic heart-murmurs, 114
Organic stricture of the esophagus, 195
Orthotonos, 347
Osteitis deformans, 357
Osteo-arthropathy, hypertrophic, pulmonary, 358
Ovary, right, abscess of, 210
Oxaluria, 248
Oxyuris vermicularis, 218

PACHYMENINGITIS, 315, 316
Pachymeningitis, cervical, 286
Pachymeningitis, cervical, differentiated from—
  progressive muscular atrophy, 286
  subacute anterior poliomyelitis, 286
Pachymeningitis, hemorrhagic, 315, 316
Pachymeningitis, hypertrophic, differentiated from—
  syringomyelia, 312
  tumor of the spinal cord, 311
Pachymeningitis, lumbar, differentiated from posterior spinal sclerosis, 301
Pachymeningitis, spinal, differentiated from chronic myelitis, 290
Pachymeningitis, spinal, differentiated from multiple neuritis, 277
Paget's disease, 357
Palpation, 110, 148
Palsy, bulbar, acute, 296
Palsy, bulbar, acute, distinguished from chronic bulbar palsy, 296
Palsy, bulbar, differentiated from pseudo-bulbar palsy, 296
Palsy, pseudo-bulbar, 296
Palsy, pseudo-bulbar, differentiated from true bulbar palsy, 296
Palsy, shaking, 306
Paludism, larval, 57
Panaris, analgesic, 312
Pancreas, 241
Pancreatitis, acute, 241
Paralysis, 272
Paralysis agitans, 306
Paralysis agitans differentiated from cerebro-spinal sclerosis, 307
Paralysis, ascending, acute, 291
Paralysis, ascending, acute, differentiated from—
  acute anterior poliomyelitis, 292
  acute myelitis, 292
  multiple neuritis, 292
Paralysis, bulbar, progressive, 295
Paralysis, cerebral, differentiated from acute anterior poliomyelitis, 291
Paralysis, general, of the insane, 332
Paralysis, general, of the insane, differentiated from posterior spinal sclerosis, 333
Paralysis, glosso-labio-laryngeal, 292, 293
Paralysis, hysterical, 346
Paralysis, Landry's, 291
Paralysis of the diaphragm differentiated from—
  asthma, 184
  paralysis of the phrenic nerve, 281
Paralysis of the facial nerve, 279
Paralysis of the musculo-spiral nerve, 281
Paralysis of the phrenic nerve, 281
Paralysis, pseudo-hypertrophic, 296
Paralysis, pseudo-hypertrophic, differentiated from—
  congenital spastic paraplegia, 320
  simple idiopathic muscular atrophy, 299

Paraplegia, 272, 273
Paraplegia, ataxic, 303
Paraplegia, ataxic, hereditary, 304
Paraplegia, ataxic, hereditary, differentiated from postero-lateral sclerosis, 304
Paraplegia, hysterical, 345
Paraplegia, spastic, 302
Paraplegia, spastic, congenital, 319
Paraplegia, spastic, congenital, differentiated from pseudo-hypertrophic paralysis, 320
Paraplegia, spastic, differentiated from hysterical paraplegia, 346
Parasites, intestinal, 214
Parasitic stomatitis, 185
Parenchymatous tonsillitis, 191
Paresis, 272
Paresthesia, 273
Paretic dementia, 332
Parkinson's disease, 306
Parotiditis, 188
Parotiditis differentiated from laryngitis, 139
Paroxysmal hemoglobinuria, 255
Paroxysmal hemoglobinuria differentiated from hemorrhagic malarial fever, 58
Pathological association, diagnosis by the method of, 20
Pectoriloquy, 152
Pectoriloquy, whispering, 152
Percussion, 110, 149
Percussion, auscultatory, 150
Percussion, immediate, 149
Percussion, mediate, 149
Percussion, respiratory, 150
Perforation of the bowel in typhoid fever, 40
Pericardial effusion differentiated from—
  dilatation of the heart, 121
  pleuritic effusion, 157
Pericarditis, acute, 127
Pericarditis, acute, differentiated from—
  acute endocarditis, 129
  acute pleurisy, 128
Perihepatitis, 235
Perihepatitis, chronic, differentiated from cirrhosis of the liver, 236
Periodic vasomotor coryza, 137
Perirenal abscess, 269
Peritonitis, acute, 219
Peritonitis, acute, differentiated from—
  acute enteritis, 220
  acute gastritis, 219
  acute intestinal obstruction, 220
  intestinal colic, 220
  subacute rheumatism, 221
Peritonitis, chronic, 221
Peritonitis, chronic, differentiated from malignant disease of the liver, 222
Perityphlitis, 209
Perityphlitis, differential diagnosis of, 210
Perityphlitis differentiated from—
  abscess of the right ovary, 210
  carcinoma of the cecum, 211
  lumbar abscess, 210
Pernicious anemia, 98
Pernicious malarial fever, 56
Pernicious malarial fever, diagnosis of, 58
Pertussis, 145
Petit mal, 342
Pharyngitis, 188
Pharyngitis, acute catarrhal, 188
Pharyngitis, acute phlegmonous, 189
Pharyngitis, acute tuberculous, 189
Pharyngitis differentiated from laryngitis, 138
Pharyngitis, gangrenous, 193
Pharyngitis, tuberculous, differentiated from—
  syphilitic sore-throat, 190
  typhoid fever, 190
Pharyngoscopy, 135
Pharynx, 188
Pharynx, herpes of, 192
Phlebitis, portal, differentiated from acute hepatitis, 225
Phlegmonous pharyngitis, acute, 189
Phosphoric acid in urine, 246
Phosphorus-poisoning differentiated from acute yellow atrophy of the liver, 227
Phrenic nerve, paralysis of, 281
Phrenic nerve, paralysis of, differentiated from—
  degeneration of the diaphragm, 281
  inflammation of the diaphragm, 281
  superior intercostal breathing, 281

Phthisis, florid, 169, 174
Physical diagnosis, 147
Physical signs, 18
Pia-arachnitis, 315
Plastic bronchitis, 159
Pleural effusion, 181, 182
Pleurisy, acute, 153
Pleurisy, acute, differentiated from—
  acute pericarditis, 128
  acute pneumonia, 154
  intercostal neuralgia, 155
Pleurisy, chronic, differentiated from—
  interstitial pneumonitis, 179
  intrathoracic tumor, 156
  pulmonary tuberculosis, 175
Pleurisy differentiated from pernicious malarial fever, 58
Pleuritic effusion, 155, 156, 157
Pleurosthotonos, 347
Plumbism, 337
Pneumonia, acute croupous or lobar, 163
Pneumonia, acute, differentiated from acute pleurisy, 154
Pneumonia, catarrhal, 162
Pneumonia, catarrhal, differentiated from—
  atelectasis, 163
  bronchiectasis, 160
  capillary bronchitis, 162
  croupous pneumonia, 166
  pulmonary infarction, 163
Pneumonia, croupous, differentiated from—
  catarrhal pneumonia, 166
  pulmonary congestion, 166
  pulmonary edema, 166
  pulmonary infarction, 167
  pulmonary tuberculosis, 167
Pneumonia differentiated from—
  pernicious malarial fever, 58
  typhoid fever, 42
Pneumonia, lobular, 162
Pneumonitis, interstitial, 179
Pneumonitis, interstitial, differentiated from—
  chronic bronchitis, 179
  chronic pleurisy, 179
Pneumothorax, 181
Pneumothorax differentiated from—
  diaphragmatic hernia, 182
  pleural effusion, 182
  pulmonary cavity, 182
  pulmonary emphysema, 180
Poikilocytes, 96
Poisoning, arsenical, 50
Poisoning, narcotic, differentiated from sunstroke, 335
Poliomyelitis, anterior, acute, 290
Poliomyelitis, anterior, acute, differentiated from—
  acute ascending paralysis, 292
  acute myelitis, 291
  cerebral paralysis, 291
  multiple neuritis, 291
  progressive muscular atrophy, 295
Poliomyelitis, anterior, subacute, differentiated from cervical pachymeningitis, 286
Polyneuritis, 276
Polyuria, 256
Polyuria, hysterical, differentiated from diabetes insipidus, 257
Portal phlebitis, 225
Posterior spinal sclerosis, 300
Postero-lateral sclerosis, 303
Previous history, 19
Primary lateral sclerosis, 302
Primary sciatica, 279
Progressive bulbar paralysis, 295
Progressive muscular atrophy, 292
Progressive paralysis of the insane, 332
Pseudo-bulbar palsy, 296
Pseudo-hydrophobia, 350
Pseudo-hypertrophic paralysis, 296
Pseudo-leukemia, 101
Pseudo-leukemia differentiated from—
  leukemia, 102
  tuberculosis of the lymph-glands, 102
Pseudo-leukemic anemia, infantile, 103
Pseudo-membranous laryngitis, 143
Pulmonary abscess, 160, 167, 168, 176
Pulmonary carcinoma, 175
Pulmonary cavity, 182
Pulmonary congestion, 166
Pulmonary edema, 166
Pulmonary emphysema, 179
Pulmonary gangrene, 168
Pulmonary hemorrhage, 175
Pulmonary hypertrophic osteo-arthropathy, 358
Pulmonary incompetency or regurgitation, 125

Pulmonary infarction, 163, 167
Pulmonary obstruction, 125
Pulmonary syphilis, 176
Pulmonary tuberculosis, 169
Pulsatory empyema, 153
Purity of the heart-sounds, 112
Purpura, 105
Purpura differentiated from—
  measles, 106
  scorbutus, 106
Purring tremor, 110
Putrid bronchitis, 159
Putrid sore-throat, 193
Pyelitis, 249, 263
Pyelitis differentiated from cystitis, 263
Pyemia differentiated from—
  acute rheumatism, 88
  typhoid fever, 40
Pylephlebitis differentiated from catarrhal jaundice, 237
Pyonephrosis, 271
Pyuria, 249

QUANTITY of urine, 242
Quinsy, 191

RACHITIS, 108
Rachitis differentiated from mollities ossium, 109
Rag-weed fever, 137
Râles, 151
Rational signs, 19
Raynaud's disease, 133
Raynaud's disease differentiated from—
  frost-bite, 134
  Morvan's disease, 314
Reaction of degeneration, 274
Reaction of urine, 243
Recrudescence in typhoid fever, 39
Reflex symptoms, 19
Reflexes, 273
Reflexes, alterations in, in disease, 274
Reflexes, deep, 274
Reflexes, superficial, 273
Regurgitation, aortic, 124
Regurgitation, mitral, 122
Regurgitation, pulmonary, 125
Regurgitation, tricuspid, 125
Relapse in typhoid fever, 39
Relapsing fever, 51
Relapsing fever differentiated from—
  typhus fever, 52
  yellow fever, 52
Remittent fever, 30
Remittent fever, malarial, 55
Renal calculus, 264
Renal colic, 239
Renal inadequacy, 263
Resonance, vocal, 152
Respiratory percussion, 150
Respiratory system, 134
Retro-pharyngeal abscess, 194
Rheumatic fever, 87
Rheumatism, 90
Rheumatism, acute, 87
Rheumatism, acute, differentiated from—
  acute gout, 92
  acute synovitis, 88
  pyemia, 88
Rheumatism, chronic, 90
Rheumatism, chronic, differentiated from rheumatoid arthritis, 95
Rheumatism of the muscles of the back differentiated from spinal meningitis, 285
Rheumatism, subacute, 89
Rheumatism, subacute, differentiated from—
  acute peritonitis, 221
  neuralgia, 89
  neuritis, 276
  trichiniasis, 90
Rheumatoid arthritis, 94
Rheumatoid arthritis differentiated from—
  chronic rheumatism, 95
  gout, 95
Rhinoscopy, 135
Risus sardonicus, 347
Rose-cold, 137
Roseola, 68
Rötheln, 68
Rötheln differentiated from—
  measles, 69
  scarlet fever, 69
Round-worms, 217
Rubeola, 63

SARCOMA of the omentum differentiated from carcinoma of the stomach, 202
Scarlatina, 65
Scarlet fever, 65

Scarlet fever differentiated from—
  dengue, 77
  diphtheria, 78
  erysipelas, 74
  measles, 68
  Rötheln, 69
  smallpox, 72
Sciatic neuralgia, 278
Sciatica, 278
Sciatica differentiated from—
  disease of the hip-joint, 278
  sciatic neuralgia, 278
Sciatica, primary, differentiated from secondary sciatica, 279
Sciatica, secondary, differentiated from primary sciatica, 279
Sclerodactylia, 314
Scleroderma differentiated from—
  Morvan's disease, 313
  myxedema, 355
Sclerosis, cerebro-spinal, 305
Sclerosis, cerebro-spinal, differentiated from—
  cerebral tumor, 331
  paralysis agitans, 307
  postero-lateral sclerosis, 305
Sclerosis, disseminated, 305
Sclerosis, insular, 305
Sclerosis, lateral, amyotrophic, 292, 293
Sclerosis, lateral, differentiated from chronic myelitis, 290
Sclerosis, lateral, primary, 302
Sclerosis, lateral, primary, differentiated from—
  cerebral hemiplegia, 303
  chronic myelitis, 302
  postero-lateral sclerosis, 304
Sclerosis, multiple, 305
Sclerosis, postero-lateral, 303
Sclerosis, postero-lateral, differentiated from—
  cerebellar tumor, 304
  cerebro-spinal sclerosis, 305
  hereditary ataxic paraplegia, 304
  posterior spinal sclerosis, 303
  primary lateral sclerosis, 304
Sclerosis, spinal, posterior, 300
Sclerosis, spinal, posterior, differentiated from—
  cerebellar tumor, 302
  general paralysis of the insane, 333
  lumbar pachymeningitis, 301
Sclerosis, spinal, posterior, differentiated from—
  multiple neuritis, 301
  postero-lateral sclerosis, 303
Scorbutus, 104
Scorbutus differentiated from purpura, 106
Scurvy, 104
Seat-worms, 218
Secondary sciatica, 279
Sensation, 273
Septic infection, fever of, differentiated from malarial fever, 57
Sequelæ, 22
Shaking palsy, 306
Ship-fever, 43
Sick headache, 284
Signs, 18
Signs, general or constitutional, 19
Signs, local, 19
Signs, negative, 23
Signs, physical, 18
Signs, rational, 19
Simple continued fever, 31
Simple idiopathic muscular atrophy, 298
Smallpox, 69
Smallpox, confluent, 70
Smallpox differentiated from—
  cerebro-spinal fever, 48
  chickenpox, 73
  erysipelas, 75
  glanders, 81
  measles, 72
  scarlet fever, 72
  typhoid fever, 42
  typhus fever, 44
Smallpox, discrete, 70
Smallpox, hemorrhagic, 71
Smallpox, malignant, 71
Sodium chloride in urine, 245
Softening, cerebral, 327
Solvent power of the gastric juice, 196
Sore-throat, common membranous, 192
Sore-throat, common membranous, differentiated from diphtheria, 192
Sore-throat, gangrenous, differentiated from diphtheria, 193
Sore-throat, herpetic, 192
Sore-throat, putrid, 193
Sore-throat, syphilitic, differentiated from tuberculous pharyngitis, 190
Spasm, 273

Spasmodic croup, 142
Spastic paraplegia, 302
Spastic paraplegia, congenital, 319
Specific fever, 30
Specific gravity of urine, 243
Sphygmogram, 115
Sphygmograph, 115
Spinal compression, 309
Spinal hemorrhage, 308
Spinal membranes, hemorrhage into, 286
Spinal meningitis, 285
Spinal pachymeningitis, 277, 290
Spinal tumor, 310
Spleen, 240
Spleen, abscess of, 156
Spleen, floating, 241
Splenic fever, 81
Splenitis, 241
Spool-worms, 218
Sporadic cretinism, 354
Sporadic cretinoidism, 354
Sputum, tubercle-bacilli in, 172
Status epilepticus, 342
Stomach, 195
Stomach, carcinoma of, 200
Stomach, dilatation of, 199
Stomach, neuralgia of, 196
Stomatitis, aphthous, 184
Stomatitis, catarrhal, 184
Stomatitis differentiated from diphtheria, 80
Stomatitis, gangrenous, 186
Stomatitis, mercurial, 186
Stomatitis, parasitic, 185
Stomatitis, ulcerative, 185
Stricture, functional, of the esophagus differentiated from organic stricture, 195
Stricture of the esophagus, 194
Strychnine-poisoning differentiated from tetanus, 348
Subacute rheumatism, 89
Subfebrile temperature, 28
Subjective symptoms, 17
Subnormal temperature, 29
Sugar in the urine, Boettger's test for the presence of, 259
Sugar in the urine, Fehling's test for the presence of, 259
Sugar in the urine, fermentation-test for the presence of, 260
Sugar in the urine, Moore's test for the presence of, 259
Sugar in the urine, Trommer's test for the presence of, 259
Sulphuric acid in urine, 246
Sun-pain, 57
Sunstroke, 32, 334
Sunstroke differentiated from—
  acute alcoholism, 335
  acute meningitis, 335
  cerebral hemorrhage, 335
  heat-exhaustion, 335
  narcotic poisoning, 335
  uremia, 268, 335
Superficial reflexes, 273
Suppuration, fever of, differentiated from malarial fever, 57
Supra-renal bodies, tuberculosis of, 107
Symmetrical gangrene, 133
Symptomatic fever, 30
Symptoms, 17, 18
Symptoms, objective, 17
Symptoms, reflex, 19
Symptoms, subjective, 17
Syncope differentiated from—
  cerebral hemorrhage, 325
  epilepsy, 343
Synovitis, acute, differentiated from acute rheumatism, 88
Synovitis, gonorrheal, 88
Syphilis differentiated from—
  glanders, 81
  Morvan's disease, 314
Syphilis, pulmonary, differentiated from pulmonary tuberculosis, 176
Syphilitic arthritis, 89
Syphilitic fever differentiated from malarial fever, 59
Syphilitic sore-throat, 190
Syringomyelia, 312
Syringomyelia differentiated from—
  chronic myelitis, 312
  hypertrophic pachymeningitis, 312
  Morvan's disease, 314
  progressive muscular atrophy, 312

TABES **dorsalis, 300**
Tabes mesenterica, 222
Tachycardia, 116
Tachycardia differentiated from—
  angina pectoris, 117
  exophthalmic goiter, 117

Tachycardia differentiated from irritable heart, 118
Tactile fremitus, 148
Tape-worm, 214
Temperature, 27
Temperature, febrile, 28
Temperature, inverse, 28
Temperature, subfebrile, 28
Temperature, subnormal, 29
Tenia mediocanellata, 215
Tenia solium, 214
Tetanus, 347
Tetanus differentiated from—
    acute spinal meningitis, 348
    cerebro-spinal fever, 47
    hemorrhage into the spinal membranes, 348
    hydrophobia, 351
    hysteria, 348
    strychnine-poisoning, 348
    tetany, 349
Tetany differentiated from tetanus, 349
Thermic fever, 32, 334
Thomsen's disease, 300
Thoracic aneurism, 131
Thread-worms, 218
Throat, diphtheroid, 192
Thrombosis, cerebral, 329
Thrombosis, cerebral, differentiated from cerebral hemorrhage, 329
Thrush, 185
Tic douloureux, 283
Tongue, 187
Tongue, black or hairy, 188
Tonsillitis, 191
Tonsillitis differentiated from—
    diphtheria, 79
    laryngitis, 139
Tonsillitis, herpetic, 192
Tonsillitis, lacunal or follicular, 191
Tonsillitis, parenchymatous, 191
Torticollis, 338
Torticollis differentiated from cerebro-spinal fever, 47
Touch, 148
Translumination, 134
Transparency of urine, 243
Tremor, purring, 110
Trichiniasis, 85, 214
Trichiniasis differentiated from—
    subacute rheumatism, 90
    typhoid fever, 42
Tricuspid incompetency or regurgitation, 125
Tricuspid obstruction, 125
Trifacial neuralgia, 283
Trigeminal neuralgia, 283
Trommer's test for the presence of sugar in the urine, 259
Tube-casts in urine, 251
Tubercle-bacilli in sputum, 172
Tuberculosis, acute miliary, 177
Tuberculosis, acute miliary, differentiated from—
    acute bronchitis, 158
    capillary bronchitis, 161
    typhoid fever, 178
Tuberculosis, chronic, of the larynx, 147
Tuberculosis of the bronchial glands differentiated from whooping-cough, 177
Tuberculosis of the kidney, 269
Tuberculosis of the lymph-glands differentiated from pseudo-leukemia, 102
Tuberculosis of the supra-renal bodies, 107
Tuberculosis, pulmonary, 169
Tuberculosis, pulmonary, differentiated from—
    bronchiectasis, 176
    chronic bronchitis, 159
    chronic pleurisy, 175
    croupous pneumonia, 167
    malarial fever, 177
    pulmonary abscess, 176
    pulmonary carcinoma, 175
    pulmonary gangrene, 169
    pulmonary syphilis, 176
Tuberculous laryngitis, acute, 140
Tuberculous meningitis, 316
Tuberculous pharyngitis, acute, 189
Tumor, cerebellar, differentiated from—
    posterior spinal sclerosis, 302
    postero-lateral sclerosis, 304
Tumor, cerebral, 330
Tumor, cerebral, differentiated from—
    anemia, 331
    cerebro-spinal sclerosis, 331
    chronic nephritis, 331
    epilepsy, 331
    hysteria, 347
    lead-poisoning, 338
Tumor, intra-thoracic, differentiated from aneurism of the aorta, 131

Tumor, intra-thoracic, differentiated from chronic pleurisy, 156
Tumor of the brain differentiated from cerebral abscess 323
Tumor of the spinal cord, 310
Tumor of the spinal cord differentiated from—
    caries of the vertebræ, 311
    chronic myelitis, 311
    hypertrophic pachymeningitis, 311
Typhlitis, 208
Typhlitis differentiated from—
    abscess of the right ovary, 210
    carcinoma of the cecum, 211
    intussusception of the bowel, 213
    lumbar abscess, 210
Typhoid condition differentiated from typhoid fever, 41
Typhoid fever, 35
Typhoid fever, abortive, 38
Typhoid fever, ambulatory or walking, 38
Typhoid fever differentiated from—
    acute catarrhal enteritis, 204
    acute dysentery, 207
    acute miliary tuberculosis, 178
    acute yellow atrophy of the liver, 227
    a typhoid condition, 41
    cerebro-spinal fever, 48
    malarial fever, 59
    measles, 65
    pneumonia, 42
    pyemia, 40
    relapsing fever, 52
    trichiniasis, 42
    tuberculous pharyngitis, 190
    typhus fever, 44
    variola, 42
    yellow fever, 41
Typhoid fever, perforation of the bowel in, 40
Typhoid fever, recrudescence in, 39
Typhoid fever, relapse in, 39
Typhus fever, 43
Typhus fever differentiated from—
    cerebro-spinal fever, 47
    typhoid fever, 44
    variola, 44
Tyrosin, 247

ULCER, gastric, 199
    Ulcer, gastric, differentiated from chronic gastritis, 200
Ulceration of the stomach differentiated from carcinoma of the stomach, 201
Ulcerative stomatitis, 185
Ulcero-membranous angina, 192
Urea in urine, 244
Uremia, 267
Uremia differentiated from—
    cerebral hemorrhage, 326
    epilepsy, 267, 343
    sunstroke, 268, 335
Ureteritis, 249
Urethritis, 249
Uric acid in urine, 244
Urine, acetone in, tests for the presence of, 260
Urine, albumin in, 250
Urine, biliary acids in, 247
Urine, biliary coloring-matter in, 246
Urine, color of, 243
Urine, constitution of, 242
Urine, diacetic acid in, tests for the presence of, 260
Urine, examination of, 242
Urine, hippuric acid in, 245
Urine, odor of, 242, 243
Urine, phosphoric acid in, 246
Urine, quantity of, 242
Urine, reaction of, 243
Urine, sodium chloride in, 245
Urine, specific gravity of, 243
Urine, sugar in, Boettger's test for the presence of, 259
Urine, sugar in, Fehling's test for the presence of, 259
Urine, sugar in fermentation-test for the presence of, 260
Urine, sugar in, Moore's test for the presence of, 259
Urine, sugar in Trommer's test for the presence of 259
Urine, sulphuric acid in, 246
Urine, transparency of, 243
Urine, tube-casts in, 251
Urine, urea in, 244
Urine, uric acid in, 244

VACCINATION, 71
    Vaccinia, 72
Valvular disease of the heart, 121

Valvular disease of the heart differentiated from acute endocarditis, 128
Varicella, 73
Variola, 69
Varioloid, 71
Varioloid differentiated from chickenpox, 73
Vertebræ, caries of, 311
Vertigo, aural, 351
Vertigo, aural, differentiated from epilepsy, 351
Vertigo, labyrinthine, 351
Vertigo, laryngeal, 142
Vesical calculus, 262
Virus, humanized, 71
Vocal fremitus, 148
Vocal resonance, 152

WALKING typhoid fever, 38
Waxy casts of the uriniferous tubules, 252
Waxy kidney, 266
Weil's disease, 62
Weil's disease differentiated from yellow fever, 63
Whispering pectoriloquy, 152
Whooping-cough, 145
Whooping-cough differentiated from—
  asthma, 184
  laryngismus stridulus, 146
  membranous croup, 145
  tuberculosis of the bronchial glands, 177
Wool-sorter's disease, 81
Writers' cramp, 339
Wry neck, 338

YELLLOW fever, 59
Yellow fever differentiated from—
  acute yellow atrophy of the liver, 61
  catarrhal jaundice, 237
  cerebro-spinal fever, 48
  hemorrhagic malarial fever, 61
  malarial remittent fever, 60
  relapsing fever, 52
  typhoid fever, 41
  Weil's disease, 63

LANE MEDICAL LIBRARY

To avoid fine, this book should be returned on or before the date last stamped below.

L73 Cohen, S.S. 88976
C67 Essentials of
1892 diagnosis.

NAME	DATE DUE